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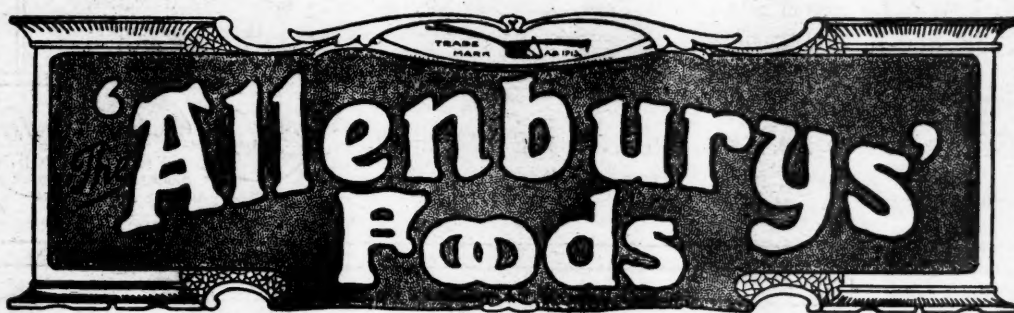
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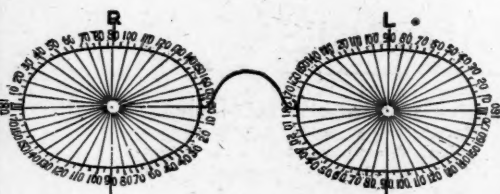
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SYDNEY: DECEMBER 9, 1916.

No. 24.

SOME OBSERVATIONS ON CEREBRO-SPINAL FEVER IN THE MILITARY CAMPS AND COUNTRY HOSPITALS OF NEW SOUTH WALES.¹

By Wolfe S. Brown, M.B., Ch.M. (Syd.),
Honorary Captain A.A.M.C., Sydney.

Introduction.

At the outset I desire to thank Professor Welsh, who has placed the entire resources of the Sydney University laboratory at the disposal of the Principal Medical Officer, Colonel Stokes. Since the July meeting I have had an opportunity of putting the therapy then outlined by us to further tests, having been deputed by the Principal Medical Officer to supervise these cases in the country camps. At the same time civil cases in the country hospitals were treated in consultation with the local practitioners.

The striking and consistent results published previously have been repeated. In all I have attended 33 patients. The cases were unselected. Some were fulminating in type. Four deaths occurred, giving a total mortality of 12%. The mortality in the 21 military cases was 9%, two patients having died. Of the four patients who died only one received intensive serum therapy, whilst of the remaining three, one died four hours after admission, having received 15 c.cm. of serum only.

The striking reduction in the death-rate under efficient serum therapy is the natural corollary of a reasoned campaign against the meningococcus. Nor is a happy result under serum therapy obtained for the first time. In the *Practitioner* of January, 1916, Gardner Robb, of Belfast, an authority on cerebro-spinal fever, writes as follows:—

Before the introduction of the serum of Flexner, in the first seven months of the 1907 outbreak, I had under my care, in the Fever Hospitals, 275 cases, and the death-rate was over 72%. In September, 1907, I began the use of Flexner's serum, and the change in results was dramatic; the death-rate fell at once to one-third of what it had been—26%. And during the same four months as the serum was used the mortality in cases outside the hospital where serum was not used was 85%.

The further reduction of death-rate (12%) I believe to be due solely to a more intensive and logical administration of the serum than that hitherto employed.

In all cases the diagnosis was supported by a bacteriological examination, the meningococcus giving the requisite tests.

Before stating the routine of treatment adopted in the meningeal stage of the disease, I wish to bring before you three factors noted which are worthy of our consideration.

(a) No less than four cases of the series had been treated in the first instance as influenza. After the patients had settled down they were discharged cured, only to be readmitted at periods varying from 2 to 14 days, with a sudden acute attack of cerebro-spinal meningitis. Some of the cases were treated for the first 12 hours of admission to hospital as

influenza cases, and observation made of the absence of meningeal signs.

(b) The second factor I have not heard mentioned before. No less than six cases were said to have supervened on measles, the rash being said to be typical. Both these factors were obviously confused cases of cerebro-spinal fever in the septicæmic stage of the disease.

(c) The third factor is a most important one from the practical standpoint. It has hitherto been accepted that lumbar puncture normally produces a fluid which drips over at less than one drop per second, and is as clear as water. I have been asked if I would regard it as an indication for thecal serum therapy whilst awaiting further tests in a case suspicious enough to warrant a diagnostic lumbar puncture, and perfectly clear fluid came over under increased pressure. My answer is no. I have punctured in at least 12 cases in which 1½ ounces and more of perfectly clear fluid under increased pressure were obtained, and have never known them to turn out to be cerebro-spinal meningitis cases. Furthermore, it may be averred that these may have been abortive cases. I am satisfied that they were not. In the first place, the naso-pharyngeal swab yielded a negative bacteriological finding in each case. Secondly, some of the cases were shown subsequently to be of other forms of infection, e.g., pneumonia. In the third place, these cases were not characterized by leucocytosis, except when other forms of inflammation were definitely present. I have often noticed that an ounce or more of cerebro-spinal fluid can often be obtained without any difficulty, when required for a diagnostic Wassermann test in other cases. Of course I am excluding septicæmic cases of the disease. If a case were regarded as suspicious a blood culture was always taken.

Treatment.

(a) Serum Therapy.

All the patients received treatment on intensive serum lines from the moment they came under observation, that is to say, lumbar puncture was carried out, and the patients received two doses of 30 c.cm. of serum into the spinal theca within the first 18 hours of coming under observation. This was done whether improvement followed or not after the first puncture. So important was this deemed in the military cases that if the first serum was given at 2 p.m. we would arise at 2 or 3 a.m. to repeat the puncture and serum. The foot of the bed was raised after injection had been carried out. Lumbar puncture was performed usually under local anaesthesia, and after the first two punctures, as often as symptoms called for it. This was at least once in the 24 hours for the first five days, and often twice or more often in the 24 hours. Serum was administered by the spinal route until a sterile cerebro-spinal fluid was obtained, and serum was occasionally given in addition by the subcutaneous route at

¹ Read at a meeting of the New South Wales Branch of the British Medical Association, on October 27, 1916.

the same time. The amount of cerebro-spinal fluid removed at each puncture was altered from that previously adopted. Instead of being guided by excessive pressure only, as evidenced by the rate of flow from the puncture needle, the plan was conceived of draining away as much of the infected fluid as possible without causing too much distress, and always getting below normal pressure. A usual amount was from 50 to 70 c.cm. During the first two punctures at least 45 c.cm. would be removed at each puncture.

It was noted, again, that in some cases, especially if the fluid were very purulent, although pressure symptoms were marked, only a slow drop could be obtained from the puncture needle. In these cases it was usual to wait until about 50 c.cm. were obtained. The head would have to be raised on a couple of pillows, and even then one to two hours would be spent in obtaining it. The local anæsthetic was an advantage in these conditions, and the patient seemed to get nothing but benefit from the above procedure.

It seemed to me more than ever that serum should be given in doses of 60 c.cm. within the first 18 hours, since at the commencement you have no indication as to the course the case is likely to take without it; but that it was injurious to keep on giving it after the cerebro-spinal fluid was sterile. If you persisted then in giving serum it seemed to delay or prevent a crisis, causing a condition of serum fever, with excessive accumulation of cerebro-spinal fluid, which remained turbid and sterile. Hence, for efficient serum therapy, culture media and an incubator were taken from case to case. A small incubator with a kerosene lamp and regulator was used. This turbid sterile fluid rapidly became clear if puncture alone were performed, and a crisis almost always occurred about the third to the seventh day.

In some cases the patients were not treated by the intensive treatment. If recovery ensued after lumbar puncture, with or without an occasional injection of 15 c.cm. of serum, the course of the disease tended to be more chronic. It is the cases that are treated in a haphazard manner that are responsible for the opinion that serum is of no use. The fluid in these more chronic cases was almost clear, opalescent, and as a rule yielded, on culture, a growth of meningococcus. These cases did not as a rule yield at once to intensive serum therapy, but if favourable ended by lysis, after some weeks. The patients seemed to derive considerable benefit from vaccine, especially if autogenous. The serum used by me was almost exclusively that of Parke, Davis and Co.

(b) *Treatment of Naso-pharynx.*

In addition to serum therapy all the patients received vigorous local treatment of the nose and naso-pharynx, chiefly by a spray of hydrogen peroxide (10 vol. %) in the acute stage, and by irrigation with hydrogen peroxide (1.3 vol. %) in the inverted position, through the nares if swabs were positive when convalescence was established.

(c) *Helmitol; Urotropine.*

In regard to drugs, all patients received either helmitol or, when unobtainable, urotropine by mouth,

in doses of 10 grains every four hours. Helmitol has been found in the alkaline cerebro-spinal fluid, and is possibly adjuvant, whilst both are of value in disinfecting the urine, which may contain the meningococcus.

Hydrocephalus.

A complication that is much spoken of in the treatment of this disease is hydrocephalus. A condition of hydrocephalus due to total blockage of the foramina in the roof of the fourth ventricle, seems to me, so far, very rarely if ever to be present. On the other hand, a condition of relative hydrocephalus, with large bulging ventricles, and relatively small spinal communication is extremely common. A not unusual condition is that after a certain number of punctures have been carried out the amount of fluid obtained gets less and less, until it escapes in slow drops. This takes place in spite of the presence of pressure symptoms, such as marked headache, vomiting, neck stiffness, even with head retraction and nystagmus. It has been suggested that these cases would be suitable ones for the draining of one or both lateral ventricles. But I am opposed to this, since lumbar puncture is still the efficient and successful means of treatment, provided that it is properly performed. It should be done as often as twice, three times, or more frequently in the 24 hours, a sufficient length of time (1 to 3 hours) being allowed to elapse for a sufficient quantity of fluid to collect. This procedure should, I submit, be given a trial before the formidable task of draining the lateral ventricle is resorted to. According to reports, the draining is rarely successful. I have had three of these cases in the series, and all the patients recovered after puncture. The condition of dry spine, on the other hand, is a totally different thing. It must be very rare indeed, and I have yet to see a case. Many cases are spoken of as instances of dry spine, but few are. In the only two cases of this kind dealt with by me while I was Pathologist at the Royal Prince Alfred Hospital, this diagnosis, made prior to a post mortem examination, proved to be incorrect.

Relapsing Cases.

The relapsing cases met with were of interest. Whatever theory is advanced as to their cause, I am more and more forced to the conclusion that reinfection from the naso-pharynx and the accessory sinuses plays an important part. It seems essential to treat the nose and naso-pharynx, etc., during the course of the disease, and to ensure that this region is free by swabbings before the patient is discharged from hospital. It has been objected that the naso-pharynx in these cases tends to become free of the organisms without any special treatment. This may be so in some cases, but a large percentage remain positive even after convalescence is well established.

Sophian, in his work, quotes the following statistics:—

Netter and Debré found the organism as follows:

- First week of disease in 78% of the cases.
- Second week of disease in 60% of the cases.
- Third week of disease in 50% of the cases.
- Fourth week of disease in 25% of the cases.

Later than fourth week of disease in 15% of the cases.

I have isolated the organisms from the naso-pharynx of a case as late as 2½ months after the commencement of the disease. I had an opportunity of examining the naso-pharynx of three out of five cases of relapse, and in all positive results were obtained. It may be objected that the naso-pharynx again becomes positive with the onset of a fresh attack of the disease, but in the following case this was certainly not so.

Case 23 (Coota).—Pte. M., after a typical course of the disease, was discharged from the district hospital as quite well after walking about in the hospital grounds for some seven days. He was taken to the camp isolation hospital for naso-pharyngeal swabbings. The swabbings, I found, contained numerous meningococci. About three days later he had an acute onset of typical meningitis, lumbar puncture yielding 54 c.cm. of very turbid fluid teeming with meningococci. He was placed under intensive serum therapy. The crisis occurred on the fourth day, and he made an uninterrupted recovery. He has since given negative swabbings, and is in training again. Relapsing cases are always treated on all fours with a primary acute stage of the disease.

Quite a different thing from relapses is a recrudescence of the disease after an apparent crisis whilst the patient is still ill with the disease. This again seemed to occur more often in those cases in which the intensive serum therapy had not been applied at the outset of the disease. It was also observed in cases in which free drainage was not obtained by adequate lumbar puncture, even if the serum had been pushed. In these cases the recrudescence was followed by crisis if puncture was carried out twice or three times, and about 50 c.cm. of serum were withdrawn each time.

Prophylaxis.

The preventive measures employed in camp consisted of:

- (a) Detection of catarrhal stages of the disease.
- (b) The elimination of carriers.
- (c) The examination of all convalescents until two consecutive negative swabbings were obtained.

Catarrhal Cases.—At the height of the epidemic all persons suffering with naso-pharyngeal catarrh were encouraged to report for treatment. A naso-pharyngeal swabbing was taken by a modified West swab, and the meningococcus sought in the culture. It was found that 5.8% were positive (119), excluding contacts. The patients were then subjected to a naso-pharyngeal wash-out with 10 vol. % hydrogen peroxide, diluted with three parts of water. The patients with catarrh and contacts were isolated until they had yielded negative swabbings. It is of interest that two of the catarrhal patients in whom positive results were obtained were:—

(a) Cpl. W., a person with deflected septum and chronic rhinitis, who, on being questioned, was traced to close contact with a patient with cerebro-spinal fever, at the Royal Prince Alfred Hospital. He was

proved to have a positive naso-pharyngeal swabbing.

(b) Pte. M., a contact of Pte. W., slept in the same tent. He subsequently developed cerebro-spinal meningitis. The examination of his naso-pharynx was positive.

The contacts were all isolated at once, and treated like catarrhal patients pending the results of the bacteriological investigation; 343 were examined; 14% were positive.

This is a fairly high percentage when it is remembered that owing to pressure of work it was often a week or more before the examination could be carried out. The contacts were isolated and under treatment in the meantime, and in some the cocci disappeared from the naso-pharynx.

NOTES ON THE TREATMENT OF 35 CASES OF CEREBRO-SPINAL MENINGITIS.¹

By R. E. Nowland, M.B. (Syd.),
Royal Prince Alfred Hospital, Sydney.

First and foremost in the treatment of cerebro-spinal meningitis is the early recognition and the immediate application of rigid measures. By early recognition is meant a diagnosis within three days from the period of invasion.

The points which I have found helpful in the early diagnosis are: a history of a "cold" or "influenza" for about a week, of pains all over the body, especially in the legs, knees and back, of vomiting appearing early, of headache, which is usually frontal, but may be occipital. Then there is stiffness of the neck. Conjunctivitis may be present, and may become purulent later on. Rhinitis and defective hearing are also common symptoms. The rash usually appears within twenty-four hours; and is commonly a purpuric eruption. It may assume other forms, viz., macular, papular, or erythematous, but there may be definite subcutaneous hæmorrhages. The position in which I have found the rash most constantly in the early stages is on the buttocks and upper eyelids. In several cases, in the very early stages, I have noticed small hæmorrhagic spots on the soft palate, analogous to the purpuric eruptions of the skin. There is usually a considerable amount of congestion of the throat, associated with hypertrophy of the tonsils. Examination of the *fundus oculi* at this stage does not usually reveal anything of diagnostic importance. Kernig's sign is of early diagnostic importance; Brudzinski's is not so constant, though it is present in about 60% of the cases. The knee jerks are usually much exaggerated, and the plantar reflex is flexor. In two early cases Babinski's sign was present. Delirium and coma may occur early, but as a rule do not appear until about the fourth day. The first step in the treatment of a case presenting the features above mentioned has been to get the patients into the open air. They do much better than when confined within wards. A lumbar puncture is carried out without delay, both to confirm the diagnosis and for its therapeutic value. The technique of lumbar puncture can be

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passed over, but one word may be devoted to the site of puncturing. I prefer to go in in the middle line, and not to the side, and usually start between the spines of the fourth and fifth lumbar vertebrae, and work upwards to the spaces between the second and third at subsequent punctures. Both local and general anaesthetics have been used; the general anaesthetic used has been ether by the open method. As the degree of anaesthesia required is very light, I never hesitate to use it, and have seen no bad results from it, though, of course, local anaesthesia is used as far as possible.

On inserting the needle the fluid usually gushes out under increased pressure. This gush is controlled by inserting the stylet. If this is not done the patient complains of unbearable headache, and there is also the risk of collapse. The fluid is usually milky at the first puncture, but may be very turbid, with a heavy deposit on standing for a short time. When the cerebro-spinal fluid has stopped running, which may occur at any time up to twenty minutes, anti-meningitic serum is introduced in to the spinal canal. Thirty cubic centimetres of serum is used, even in children, provided of course that 30 c.cm. or more of fluid have been removed. The serum may be introduced either by the gravitation method or by injecting it with the syringes supplied. I have employed the latter method in all cases. It must be done slowly; from seven to ten minutes is occupied in the injection. After the serum has been injected the foot of the bed is elevated on blocks about 18 inches high, and the patient is left in this position for about six hours, during which time he usually complains of very severe headache and pains in the back and legs. Morphine will sometimes ease the pains, but if it does not within about three or four hours it is repeated, and the blocks are removed from the foot of the bed. The use of morphine should be watched, as it tends to increase the delirium if given too frequently, as one is wont to do in efforts to control the sufferings. In twelve hours another puncture is performed. Sometimes it will be necessary to puncture even before the twelve hours have elapsed, in cases where morphine fails to control the sufferings and restlessness. A second dose of serum is given at the end of 24 hours, and a third at the end of 48 hours, a lumbar puncture having been carried out at the end of 36 hours. Usually three doses of 30 c.cm. of serum is sufficient. Indication for using more are:—

- (1) No abatement of symptoms at the end of 72 hours.
- (2) The presence of meningococci in the fluid. A specimen from every lumbar puncture has been cultured, and slides made. This of course makes a pathological laboratory indispensable in the proper treatment of meningitis.
- (3) The fact that the cerebro-spinal fluid remains very turbid, not waiting for a pathological report.
- (4) In some cases where you get a great increase in the amount of the cerebro-spinal fluid.

Usually about one or two doses of serum (intrathecal) is sufficient in these cases.

In a case in which the toxæmia was general, and the condition very grave, I saw an excellent result follow the intravenous injection of 30 c.cm. of serum with normal saline solution, although the immediate effects were very alarming.

In cases with marked skin eruptions two or three doses of serum, given subcutaneously, at intervals of 24 hours, are certainly of value, both for the skin eruption and the general toxæmia. One case in particular with very marked subcutaneous hæmorrhages, responded excellently, both as regards the skin affection and the general condition. The serum which has been found most efficacious in the cases I have treated is that prepared by Parke, Davis and Co. Other serum was tried, but none gave such good results. After the patient has been given the requisite amount of serum the next question is how many times a day is lumbar puncture to be carried out. I have found the following points helpful:—

- (1) The amount of fluid obtained at each puncture when a puncture in the morning yields 50 to 60 c.cm. of fluid, and the same quantity may be obtained in about 12 hours may be less than when lumbar puncture is carried out more frequently, e.g., three times a day.
- (2) The pressure under which the fluid flows: This of course is proportional to the amount of fluid obtained.
- (3) Marked restlessness, headache, and pains in the back and legs call for frequent punctures; in one case I carried out the punctures every six hours, and for part of the time a continuous drain was left in.
- (4) The character of the fluid in regard to turbidity: If the fluid is very turbid at least two punctures should be carried out in the 24 hours. As the fluid becomes clearer fewer punctures are required.

In the straight-forward cases two punctures were usually done daily for four to five days, then one daily for about 7 to 10 days, and then at intervals of 1 to 2, 2 to 3, 3 to 4 days until the fluid became normal in colour, and only under slightly increased pressure, so that only 40 to 50 c.cm. were obtained in about 20 minutes.

The changes noticed in the colour of the fluid are interesting. In cases running a satisfactory course the turbidity becomes less in about 36 hours after the last dose of serum; then it becomes cloudy, then a golden tinge appears in the cloudiness, and then it becomes a clear gold colour. The golden tinge gradually diminishes at each subsequent lumbar puncture, until at the end of 10 days the fluid is quite normal in colour. The patients have been kept in bed for about three weeks after the fluid has become normal, having had one or two punctures in the meantime. They have then been allowed to get about very gradually, a close watch being kept over them for another three weeks, for the event of relapses. One case I saw relapse after the patient had been up and about for about 10 days, the second

attack being quite like the primary attack; he was treated accordingly. He is now convalescent. Two other cases I saw relapse, both in little boys, one three days, the other five days after the cerebro-spinal fluid had become normal and all symptoms had subsided. Both children are now quite well. I must say this is quite contrary to my observations. This is exemplified in one case in particular, viz., one of the little boys mentioned above. This patient I punctured frequently and gave no serum for about five days. When I say frequently I mean it was done every six or eight hours, and there were no indications of improvement. For three periods of about 12 hours each during the five days I left a continuous drain in his back. At the end of the five days he began to show cerebral symptoms—marked restlessness, delirium, etc. I then gave him two doses of 15 c.cm. of serum intrathecally, with intervals of 24 hours between each dose. Only 15 c.cm. were given, because the supply of serum was almost exhausted. He said that he felt easier 24 hours after the second dose; the fluid diminished in amount; it has now become normal, and he is very well. The use of urotropine is, I think, beneficial in the acute cases. It has been given to all the cases in doses of 10 grains every 4 to 6 hours, and, thanks to Dr. Chapman, formalin has been detected in the cerebro-spinal fluids of the majority of the cases, especially in those cases which have progressed satisfactorily. The amount detected is very minute from 1 in 100,000 to 1 in 200,000. In one case that relapsed it was not detected during the primary attack, but was found in the second, although the serum was given in the same doses in both attacks. The progress in the second attack was much more satisfactory than in the first, and the fluid and symptoms cleared up much more quickly.

Naso-pharyngeal sprays of eusol and glycerine in equal parts have been used with good results in all cases. The eusol and glycerine are mixed just before use. In this way the active hypochlorous acid gas is given off in the throat, and the spray is not inert as it would be if mixed in bulk. De Vilbiss's atomizer is used for spraying. Cases in which acute hydrocephalus develops seem to be hopeless; at any rate, that has been my experience. Three cases were trephined and the lateral ventricles drained, but all the patients died, one nine hours, one five days, and the third nine days after the operation. Complications, such as arthritis, panophthalmitis, orchitis, epididymitis, etc., have been treated on usual lines.

Chronic and subacute cases run a different course under treatment. In these cases the symptoms are chiefly spinal, that is, severe pains in the back and legs, stiffness and weakness of the legs, etc. Headache is usually present, but subsides. I have seen six such cases with histories of illness varying from one week to four months. All the patients have done satisfactorily. In these cases the cerebro-spinal fluid usually dries up quickly, but gradually. This drying up has not the same significance as it would have in an acute case, in which it would be one of the first signs pointing to hydrocephalus. These chronic cases have done well on polyvalent vaccines, with an

initial dose of 100 millions and subsequent doses every fifth or sixth day of double the quantity last injected up to 2,000 or 3,000 millions. During the three months three children were sent into hospital with symptoms very suggestive of meningitis, including vomiting, headache and stiffness of the neck. In these cases lumbar puncture was performed on admission, and clear fluid was obtained under high pressure. The symptoms cleared up within three or four days, and as far as I know the patients are quite well now. These cases were diagnosed as meningism, but no exciting cause, such as pneumonia or gastro-enteritis, was present. I have made it a rule never to give anti-meningitic serum in cases with clear cerebro-spinal fluid, but always to give it without waiting for a pathological report when the fluid presents any turbidity.

I have found that the chronic form of meningitis occurs more commonly in women. Females do not respond so well to treatment, even in the acute forms. There have been eleven females in the series, five of whom have died. These five, however, were practically hopeless on admission. One was ill five days before being sent into hospital, was delirious on admission, and died of broncho-pneumonia after six days' treatment. Another was a puerperal woman, who was quite unconscious on admission, and died three days later of septicæmia. Another was a woman at full term pregnancy; she died five days after admission of puerperal eclampsia. This case is interesting because the brain cord and meninges appeared to be normal post mortem. Clinically she was certainly suffering from meningitis; turbid fluid under high pressure was obtained at all the punctures. Labour was induced, and the child delivered with forceps. The infant left hospital quite well.

Another case was in a child sick for two weeks and unconscious for three days before admission. A suppurative otitis-media, mastoiditis and hydrocephalus on one side were discovered post mortem. The other side had been relieved at an operation eight days previously. Thick pus was found at the base of the brain.

The fifth patient was unconscious for 24 hours before admission, and died three days later.

As will be seen from the above, the treatment of acute cerebro-spinal meningitis calls for the unremitting attention of the medical man in charge.

As to the prophylaxis of acute cerebro-spinal meningitis, I do not intend to say much. The infectivity is low, and therefore with reasonable precautions there is not much risk of contracting the disease to the attendants on patients suffering from it. The nasal and naso-pharyngeal secretions are infectious, as also are the secretions from the eyes. In one case meningococci were isolated from a purulent conjunctivitis. I have noted that the disease attacks people in a weakly state of health, for example, puerperal and pregnant women. The majority of soldiers whom I have treated, and many of the civilians too, have come from the country. This suggests to me that city people have acquired an immunity to

the meningococcus, which country people have not had the opportunity of doing, owing to the fact that they do not come into such close contact with the disease, and that the country is not so thickly populated. Therefore in my mind it is the country soldiers who should be watched for symptoms of meningitis when in camp.

In placing these notes before you I beg to thank Drs. Rennie, Mills, Sinclair-Gillies and Fairfax for permission to refer to their cases, and Professor Welsh for his direction of the pathological aspect of the treatment, and Drs. Marjory Little and Dorothy Voss for their care in the pathological work.

SOME NOTES ON THE ADMINISTRATIVE AND EPIDEMIOLOGICAL ASPECTS OF CEREBRO-SPINAL FEVER.¹

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As far as I can ascertain, deaths from cerebro-spinal fever first began to be separately recorded in New South Wales in the year 1896, and since then no year has passed without deaths being recorded from the same cause. But the mortality has never bulked largely in the returns until the year 1915.

For many reasons the records of the earlier years cannot be considered as entirely reliable. Diagnosis without the aid of bacterioscopic methods must often have been at fault, and even when this was correct the system of nomenclature and classification adopted until more recent years appears to have been one which did not conduce to very great accuracy as regards the record of mortality from this particular disease. The list of deaths in New South Wales which is included in these notes was kindly furnished by the Government Statistician. It shows the deaths recorded from cerebro-spinal fever separately, and in another column the deaths from simple meningitis. It may be considered as probable that some of the deaths under the latter heading were due to the specific infection.

In the year 1915 the prevalence of cerebro-spinal fever increased greatly. Cases began to appear in the military camps in March, and with the advent of winter became rather numerous. On October 11 of that year cerebro-spinal fever was proclaimed a notifiable disease.

The accompanying tables give the results of twelve months' notification. The total number of persons notified during the year as suffering from cerebro-spinal fever in New South Wales has been 264, of whom 48 were soldiers occupying military camps. The remainder were civilians. The number of deaths recorded was 90, which corresponds to a case fatality of 34% of the cases notified.

As a rule, cerebro-spinal fever occurs sporadically. Even during epidemic outbreaks the incidence of the disease tends to be sporadic. In the New York epidemic of 1904 Bolduan records that out of 1,500 cases there were only 58 instances in which more than one person was affected in one house. The

rarity of infection among nurses, doctors, etc., is well recognized. As is probably also the case with diphtheria and certainly with pneumonia, the number of carriers of the meningococcus is always many times larger than the number of persons suffering from cerebro-spinal fever. It is therefore evident that only a minority of the individuals of a given population is susceptible to the disease, though most may at some time become carriers. These facts render the source of infection in any given case extremely difficult to trace, since a whole chain of carriers may have been interposed between any two given cases of cerebro-spinal fever. The most conspicuous epidemics have occurred in the United States and Germany; but other countries have been affected, and in 1915 important outbreaks occurred among soldiers in the military camps in Britain. These outbreaks have led to the production of a mass of literature, and have instigated the performance of a great deal of useful work among bacteriologists and epidemiologists and the clearing up of many obscure points in the natural history of the disease.

The most important practical problem to the epidemiologist connected with meningitis is that of the spread of infection. The meningococcus appears to have certain properties which make it improbable that the organism can maintain a saprophytic existence outside the human body. It is extremely susceptible to drying and to extremes of temperature. It has never been isolated from dust, air, or fomites, and the difficulty of recovering it from swabs which have been kept even a few hours before sowing is well known. It is, moreover, most susceptible to the action of disinfectants. Weichselbaum found that carbolic acid diluted to 1 in 800 prevents growth, and formalin diluted to 1 in 22,500 rapidly kills it.

The above facts afford some very strong reasons for believing that the infection of meningitis is not spread by means of fomites or through place infection. They are all in favour of a method of spread which is direct from person to person; and the facts on the epidemiological side entirely support the bacteriological view. Foster and Gaskell consider that rapidly changing populations are specially liable to invasion. This idea appears reasonable, on the ground that there seems to be in any given community a certain number of persons who harbour the meningococcus in their naso-pharyngeal spaces, and that when the population is rapidly changing there are constant opportunities for fresh infections to be introduced. It may be that individuals who are immune to one particular strain of the meningococcus are susceptible to another, and the constant reintroduction of fresh strains is at length successful in reinforcing the attack by a strain which is virulent to those who have been hitherto able to resist.

The military camp, in which the conditions of overcrowding during sleeping-hours and of rapid changes in the component individuals are more marked than in any other human habitation, appears both in Europe and in Australia to be specially subject to invasion. Our experience in New South Wales is very emphatic in the correlation of cerebro-spinal fever with military camps. Sydney, Liverpool, Goul-

¹ Read at a Meeting of the New South Wales Branch of the British Medical Association on October 27, 1916.

burn, Cootamundra, Bathurst, Dubbo, Kiama, are all sites of military camps, and these towns furnished 184 cases of the disease, or 70% of all the cases which occurred in New South Wales during the year of notification.

The opinion is growing that prophylaxis in cerebro-spinal fever is to be based upon the recognition and suitable treatment of the "carrier," who is regarded, and probably with justice, as being the chief, if not the only, means by which infection is spread. In military camps throughout Australia, the occupants of tents and huts in which cases of cerebro-spinal fever have appeared have been rather rigidly isolated and not allowed to return to normal conditions until their throats have been declared free from suspicious organisms. An attempt has been made in the State of Victoria to enforce corresponding precautions in civil life.

But the problem of applying very stringent treatment of "contacts" in ordinary civil populations is a different and much more onerous one than the same problem in a military camp. It has not hitherto been faced administratively by public health authorities, even in connexion with typhoid fever and diphtheria, and in this State at least special legislation would be necessary to enable health authorities to deal with it.

Apparently only a very small proportion of those persons who have been even in intimate contact with a person suffering from cerebro-spinal fever ever become "carriers." Out of 244 swabs examined by the Department of Public Health and taken from the throats of persons known to have been in close contact with patients, only 9, or 3.6%, proved to be positive. This is in fairly close accordance with what has been observed elsewhere. The whole question is complicated by the recorded fact that persons who are not known to have been in contact with a case may nevertheless harbour meningococci. In 1910, 9,000 troops who formed the garrison at Munich were examined, when it was found that 2% of them were meningococcus carriers. No cases of cerebro-spinal fever had occurred recently in the garrison when this examination was made. Eastwood, in 1915, found that 10% of 480 out-patients at St. Bartholomew's Hospital harboured in their throats organisms which resembled meningococci microscopically, culturally and in fermentation tests; and several other series of observations are recorded in which the meningococcus was found in a proportion of the throats of non-contact populations.

If "carriers" are as numerous among the general population as these facts seem to indicate, the value of stringent isolation of contacts is evidently much depreciated, and it becomes questionable whether health authorities would be warranted in enforcing it as a routine measure in ordinary civil populations. In military camps and in institutions, and even under special circumstances in civil life, there are good and weighty reasons for doing so. Take such a case as occurred a few months ago in the south of New South Wales. A shearers was attacked by cerebro-spinal fever in a shed which was just "cutting out." The contact between the sick shearers and his thirty mates in the shed had been rather close, and, as far as could be ascertained, general. Was

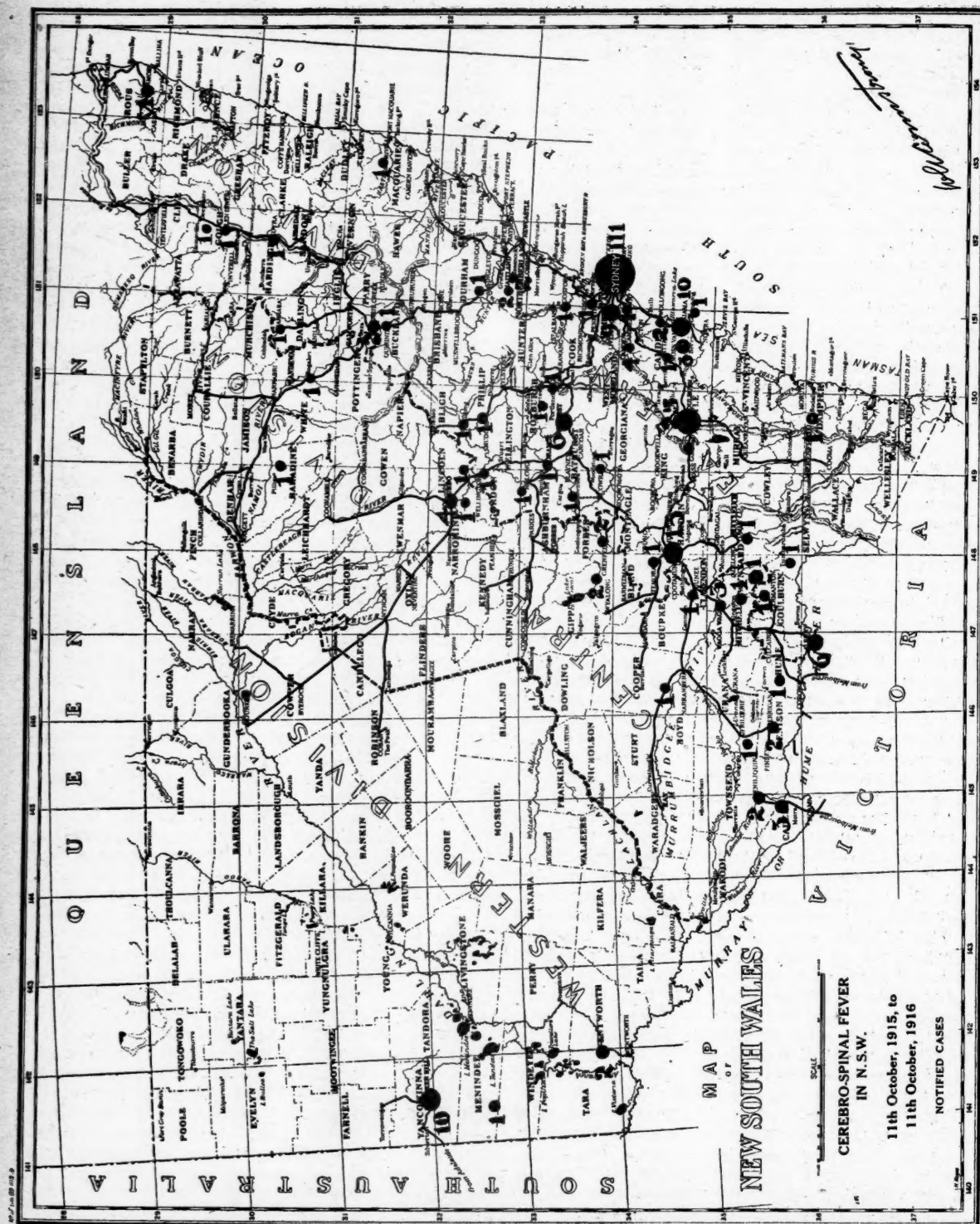
one warranted in inconveniencing all the other shearers by isolating them in the shed for three or four days while a bacteriological examination was made of all their throats, and in further isolating for a fortnight any who proved to be "carriers"? I do not think so. It was not done in this particular instance. The shearers were allowed to disperse (they could not legally have been hindered), and as no further cases were reported in that part of the country it is presumed that no evil results followed.

To a certain extent the behaviour of cerebro-spinal fever resembles that of pneumonia epidemiologically. There are known to be large numbers of carriers of pneumococci, but no one proposes to isolate them, nor would it come within the range of practical hygienics to do so.

A practical question which requires consideration is whether it is possible by therapeutic measures to free the throat of a meningococcus carrier of organisms. This appears to be very problematical with any drug hitherto tried. The application to the throat of reagents having the necessary antiseptic and penetrative power is intolerable by the patient. Recent observations by Lieutenant-Colonel M. H. Gordon at the Central Cerebro-spinal Fever Laboratory in England seem to show that a spray of the new antiseptic "chloramine" can be well tolerated by human beings, and is practically successful in effectively and rapidly ridding the throats of those exposed to it of the meningococcus. If this treatment fulfils the hopes of the discoverer it will materially facilitate the solution of the problem of the treatment of meningococcus carriers, and, incidentally, perhaps of diphtheria carriers also. A trial is to be given to the treatment by the Public Health Department of this State.

In the meanwhile, what precautions should be taken to prevent the spread of infection by means of contacts? In military camps and institutions, where rules can be stringently enforced and populations are continually under some form of discipline, the right course appears to consist in the bacteriological examination of the throats of all contacts and the rigid isolation of those who are found to be harbouring the meningococcus until they have ceased to be carriers. Local treatment of the throat of carriers with some mild antiseptic as a nasal douche is to be recommended, even if only as a placebo. Foster and Gaskell advise the use of a 1 in 1,000 solution of permanganate of potash in a 1.5% sodium sulphate solution; but they admit that it only has a transient action in clearing the throat of meningococci. It is very questionable if the permanent disappearance of the meningococcus has ever been hastened by these means and the use of stronger antiseptics is of doubtful benefit, and is accompanied by the danger of damage to the delicate mucous membrane of the naso-pharynx. In most cases carriers appear to cease to harbour the meningococcus after about a fortnight.

In civil practice, under existing legal conditions in New South Wales, it is impossible, as I have already indicated, to enforce isolation of contacts, even if they are known to be carriers. The only exception to this is that children who have been living in an



infected house must not return to school for six weeks unless they are certified by a medical practitioner to be free from infection. The medical attendant is at liberty, however, to advise the friends of the patient, and I think it is incumbent upon him to do so. I suggest as a good working rule that such persons as have been contacts of a case of cerebro-spinal fever be advised to avoid, as far as possible, coming into close relationship with other persons inside closed rooms for a few days—say a fortnight—and, of course, to avoid such close contact as is implied by kissing, to abstain also from coughing or speaking directly into another person's face, etc. In the open air there appears to be very little danger of the transference of infection, even in ordinary close association with other people.

Table I.

Showing the Number of Persons Ascertained to be Suffering from Cerebro-spinal Fever During Each Week from October 11, 1915, to October 11, 1916.

1915—	Cases.
Cases recorded to October 16	1
Cases recorded to October 23	3
Cases recorded to October 30	2
Cases recorded to November 6	2
Cases recorded to November 13	5
Cases recorded to November 20	4
Cases recorded to November 27	4
Cases recorded to December 4	2
Cases recorded to December 11	2
Cases recorded to December 18	1
Cases recorded to December 25	3
Cases recorded to December 31	1
1916—	
Cases recorded to January 1	0
Cases recorded to January 8	1
Cases recorded to January 15	1
Cases recorded to January 22	2
Cases recorded to January 29	2
Cases recorded to February 5	1
Cases recorded to February 12	1
Cases recorded to February 19	2
Cases recorded to February 26	0
Cases recorded to March 4	1
Cases recorded to March 11	1
Cases recorded to March 18	0
Cases recorded to March 25	2
Cases recorded to April 1	1
Cases recorded to April 8	1
Cases recorded to April 15	5
Cases recorded to April 22	-2
Cases recorded to April 29	2
Cases recorded to May 6	1
Cases recorded to May 13	5
Cases recorded to May 20	2
Cases recorded to May 27	7
Cases recorded to June 3	3
Cases recorded to June 10	4
Cases recorded to June 17	6
Cases recorded to June 24	4
Cases recorded to July 1	1
Cases recorded to July 8	5
Cases recorded to July 15	4
Cases recorded to July 22	8
Cases recorded to July 29	13
Cases recorded to August 5	16
Cases recorded to August 12	24
Cases recorded to August 19	10
Cases recorded to August 26	17
Cases recorded to September 2	14
Cases recorded to September 9	12
Cases recorded to September 16	14

Cases recorded to September 23	7
Cases recorded to September 30	20
Cases recorded to October 7	9
Cases recorded to October 11	3
Total	264

Table II.

Showing Number of Cases of Cerebro-spinal Fever Notified in the Metropolitan District During the Period October 11, 1915, to October 11, 1916.

Municipalities.	Cases.
City of Sydney	16
Alexandria	1
Annandale	3
Ashfield	2
Auburn	2
Balmain	2
Bankstown	1
Bexley	4
Botany	1
Canterbury	2
Cabramatta and Canley Vale	1
Drummoyne	3
Enfield	1
Glebe	3
Granville	1
Hunter's Hill	1
Hurstville	4
Kogarah	8
Lane Cove	1
Leichhardt	3
Manly	2
Marrickville	11
Mascot	1
Newtown	4
North Sydney	3
Paddington	4
Petersham	3
Randwick	5
Redfern	5
Rockdale	4
St. Peters	1
Waterloo	2
Port Jackson	1
Hornsby Shire	2
Total	108

Table III.

Showing Number of Cases of Cerebro-spinal Fever Notified in Country Municipalities During the Period October 11, 1915, to October 11, 1916.

Municipalities.	Cases.
Albury	6
Barraba	1
Bathurst	6
Bowral	1
Broken Hill	10
Campbelltown	4
Cootamundra	13
Cowra	1
Culcairn	1
Deniliquin	2
Dubbo	4
Gerrington	1
Glen Innes	1
Goulburn	25
Grenfell	2
Gulgong	1
Jamberoo	3
Junee	1
Kiama	10
Lismore	1
Liverpool	12
Mudgee	1
Orange	1
Penrith	1
Quirindi	1
Wagga	3
Wellington	1

Wentworth	2
West Maitland	2
Wollongong	2
Total	120

Table IV.

Showing the Number of Cases of Cerebro-spinal Fever Notified in Localities Outside Municipalities During the Period October 11, 1915, to October 11, 1916.

Municipalities.	Cases.
Amaroo Shire	1
Berrigan Shire	2
Blaxland Shire	2
Boggabri	1
Boree Shire	1
Cessnock Shire	2
Cobbarah Shire	1
Erina Shire	1
Eurobodalla Shire	1
Gadara Shire	1
Glennie's Creek	1
Gunning Shire	1
Guyra Shire	1
Hastings Shire	1
Holbrook Shire	1
Hume Shire	1
Kyeamba Shire	1
Menindie Police District	1
Murray Shire	3
Namoi Shire	1
Severn Shire	1
Sutherland Shire	1
Stockinbingal	1
Talbragar Shire	1
Tumbarumba Shire	1
Weddin Shire	2
Wunnamurra Shire	1
Werris Creek	2
Yanko	1
Total	36

Table V.

Showing Cases and Deaths of Cerebro-spinal Fever Notified in Military Camps During the Period October 11, 1915, to October 11, 1916.

Military Camp.	Cases.	Deaths.
Barroul	1	—
Bathurst	5	1
Cootamundra	9	2
Dubbo	4	2
Goulburn	10	3
Kiama	2	1
Liverpool	12	6
Menangle	3	1
Rutherford	1	—
Show Ground, Sydney	1	—
Totals	48	16

Table VI.

Showing Sex and Age Groups of Cases of Cerebro-spinal Fever Notified in New South Wales During the Period October 11, 1915, to October 11, 1916.

Age Groups.	Cases.		Deaths.	
	Male.	Female.	Male.	Female.
Under 6 months	2	1	3	1
6-12 months	1	7	1	2
1-2 years	6	4	2	1
2-3 years	6	9	3	4
3-4 years	6	4	4	2
4-5 years	4	6	2	—
5-10 years	16	13	3	7
10-15 years	20	9	3	5
15-20 years	40	9	8	2
20-25 years	27	7	11	2
25-35 years	24	9	7	5

35-45 years	11	5	3	2
45-55 years	4	3	—	1
55 and over	4	3	2	2
Age not stated	3	1	—	—
Totals	174	90	54	36
	264		90	

Table VII.

Showing Deaths from Meningitis and Cerebro-spinal Meningitis in New South Wales from 1880 to 1915.

Year.	Meningitis.	Cerebro-spinal Meningitis.
1880	173	—
1881	189	—
1882	169	—
1883	213	—
1884	260	—
1885	340	—
1886	231	—
1887	208	—
1888	225	—
1889	186	—
1890	176	—
1891	215	—
1892	196	—
1893	218	—
1894	223	—
1895	171	—
1896	189	18
1897	206	5
1898	202	10
1899	169	13
1900	200	26
1901	182	12
1902	177	21
1903	165	22
1904	144	39
1905	111	23
1906	124	29
1907	136	21
1908	127	24
1909	123	24
1910	132	33
1911	129	28
1912	137	30
1913	159	38
1914	161	24
1915	138	81

SOME ASPECTS OF THE ÆTIOLOGY AND EPIDEMIOLOGY OF CEREBRO-SPINAL FEVER.¹

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In considering the recent outbreaks of cerebro-spinal fever in various parts of the world, it is very important to try to ascertain with the information that is forthcoming the cause of these epidemic outbreaks of an endemic disease. Three aspects present themselves for consideration, and evidence supporting one or other of them may be exceedingly helpful in guiding our efforts at controlling the disease.

The occurrence of the present widely-separated epidemics of the disease (1) may be due to a strain of the organism in one particular locality developing an increased virulence for man and having been distributed from this source to the other affected areas, or (2) the almost contemporaneous appearance of epidemics of the disease in various parts of the world may be due to an increased virulence for man of the local strains in the affected areas, or (3)

¹ Read at a Meeting of the New South Wales Branch of the British Medical Association on October 27, 1916.

special conditions affecting man and rendering him more susceptible to the responsible organism may have arisen in each locality, thereby enabling the local strain of organism, which is responsible for a few cases of cerebro-spinal fever annually to produce a larger crop of cases than usual.

In reviewing these three possibilities it will be recognized that if the increased prevalence of the disease may in part at any rate be referred to the last-mentioned of the three, then it may be possible to take measures to reduce this incidence by reversing the process responsible for the increase. If the second factor is one in great part responsible, it will be recognized that strict measures of a quarantine type operating on incomers from another epidemic area would not necessarily have prevented the development of the disease where the complaint was already endemic, as might be the case if the first hypothesis were correct.

Has the Epidemic Disease Spread from one Centre or Developed in Several Areas Almost Contemporaneously from the Endemic Foci of the Disease?

In Australia, endemic cases of cerebro-spinal fever have been recognized for many years. The first cases recorded, presumably of this disease, were in 1873. A number of sporadic cases were recorded up to 1900. Beginning in July, 1900, there was a mild epidemic in Sydney; in 1901, in Port Pirie, in South Australia, the complaint appeared in a mild epidemic form again in the middle of July or early in August. From this period onward till the beginning of the present epidemic, occasional cases of cerebro-spinal fever have occurred annually, probably in all the capital cities of Australia. The number of endemic cases thus occurring may be gathered from the annual death returns under the heading "cerebro-spinal fever" in the metropolitan district of Sydney. These deaths vary from about 8 to 15. The disease has therefore been endemic in Australia for a number of years.

In 1900 the number of cases in Sydney slightly increased, so as to result in a mild local epidemic with a mortality of about 38 cases for the year. In the following year, in South Australia, a small epidemic occurred.

The recent epidemic in New South Wales was ushered in by a military case of meningitis occurring on March 5, another case which was fatal on May 23, another fatal one on June 1; the fourth case occurred on June 26, and on July 29 the real epidemic began by one case, followed on August 2 by three cases, and thereafter by varying numbers.

Fairley and Stewart¹ state that the Victorian epidemic of 1915 commenced vaguely, and that there were a number of isolated cases up to the end of June; but in July the numbers began to increase, especially at the military camp at Seymour.

In New Zealand Champaloup and Bowie² say that up to July 11, 1915, there had been fifteen cases, with four deaths, at Trentham Military Camp. The disease apparently began in New Zealand in June. They mention that between the years 1908 and 1913 inclusive there was an average of 125 deaths per annum in New Zealand from "simple meningitis." How many of these were due to the meningo-

coccus cannot be known. It is almost certain that a number of them were due to this organism. The meningococcus itself was definitely identified in two infants in the Dunedin Hospital in 1912.

Following the outbreaks in New Zealand, New South Wales and Victoria, there were similar slight increases in the disease in several of the other States.

From the above data it appears that in New Zealand, Victoria and New South Wales the number of cases of meningitis began to increase in June, and that this was followed in the course of a few weeks by such a number of cases that special attention was directed to their occurrence.

At the period when the disease was increasing in Australia, it was also known to exist in Egypt and in Britain. In Egypt, Chalmers and O'Farrell³ give statistics of the Egyptian army to show that there were a few cases of the disease annually up till 1913. In 1914 there were 19 cases and in 1915 52.

Did the Australian Epidemics Arise from Infections Coming from Egypt or from Britain?

Our first troops left for Egypt in November, 1914, and there they remained until the landing at Gallipoli on April 25, 1915. If Australia became infected from Egypt or Britain, the infection must have reached here before, say, the end of May. It is probable that up to this date a certain number of invalided men and disciplinary cases were returned from Egypt, and might possibly have brought back infection with them. Their numbers, however, were relatively small, and their distribution when they reached Australia would not be likely to bring them into very close touch with the troops in camp. The almost contemporaneous appearance of the disease in Victoria, New South Wales and New Zealand can hardly be explained by one or more of these places being infected from the other, unless the manifestation of the disease was so dependent on weather conditions that, after heavy seeding of the community with the organisms, favourable early spring weather had to be awaited before the disease could manifest itself. Though, of course, it is possible that a single infected individual coming from Egypt and journeying to New Zealand might infect Victoria and then New South Wales, and then New Zealand, and thus account for the almost contemporaneous outbreaks, there is no evidence of this having actually taken place, or that the disease is so intensely infective as this would indicate.

In response to an enquiry, General Fetherston has very kindly informed me that, up to the end of June, 1915, only 1,172 troops of all ranks, including 882 invalids, had returned to Australia. He has also very kindly made enquiries as to whether there was any likelihood of the disease having been introduced from Egypt, and he informs me that the early military cases were all from local camps, and were men who had only been a few days in these camps. This was also borne out in a great majority of subsequent cases. He adds that Captain Holmes, who investigated the Victorian cases and the cause of the epidemic, never had the slightest suspicion that the disease had been brought from Egypt. He concludes by adding that some men had been returned to Australia before the first case arose, but not many.

If Egypt therefore is unlikely to have been the source of infection of Australia and New Zealand, still less are the English outbreaks likely to have been the cause, inasmuch as there was a still smaller interchange of possible carriers between Britain and Australia.

We therefore return now to the possibility of the Australian and New Zealand epidemics being due to an increased virulence assumed by the strain of organism in these localities, the same increase of virulence being also shown by strains elsewhere, as, for instance, in Egypt and in Britain. There does seem definite evidence that various infectious diseases do vary in virulence from time to time for reasons unknown. It is also possible that various strains of an organism which have had a common origin, even though isolated for many years, may all independently assume increased virulence at or about the same time due to some innate quality producing cyclical periods of increased virulence. This somewhat hypothetical view may possibly play a part in originating the present epidemics.

Special Conditions Affecting Man.

One of the most important factors, however, in originating epidemics of disease seems to be certain social factors together with the season of the year. It is a most remarkable fact that cerebro-spinal fever essentially occurs during early spring to early summer. Why does it do so? Is it because the means of spread of the organism from individual to individual are more easily achieved under these climatic conditions, or is it that the individuals affected are more susceptible at these periods of the year? Our social habits do not materially differ in springtime from those in autumn. It cannot therefore be a change of habit in spring which makes human beings more liable to the disease. Similarly, the means of transmission would be presumably as effective in autumn as in spring. We are therefore reduced to the consideration that it is some particular susceptibility of the individual in spring which makes him more liable to succumb to infection at this period of the year.

We now know by examination of carriers that many individuals harbour the organism, but relatively few show symptoms of the disease. It seems probable during spring that a greater percentage of these strains of the organism affect their victims owing to greater susceptibility than is the case in other periods of the year.

But one of the most important factors in the spread of the disease, and one which has been recognized for many years, is its special prevalence under certain conditions, when men are associated together in intimate contact, as in barracks and military encampments. Strange to say, the disease does not generally appear in lunatic asylums, factories and schools where a number of individuals are in similar close contact. There is some factor therefore in military encampments which makes the individuals more susceptible to the disease, though not necessarily more susceptible to infection. In Australia and New Zealand it is clear that the number of cases of the disease amongst the military encamp-

ments has been out of proportion to the number of cases in the rest of the civil population. It is very interesting in this connexion to note that in the small epidemic in South Australia in 1901 several cases occurred among the workmen at the Bundaleer Water Works, the workmen presumably living under conditions of camp life almost similar to those experienced amongst soldiers. A factor therefore tending to favour the incidence of the disease exists in military camps and similar congregations of men, but does not usually occur in prisons, hospitals for the insane, schools or factories. What factor or factors are responsible for this difference? In military camps, new arrivals are continually coming and drafts being sent away. There is therefore a relatively rapid change in the population, presumably favouring rapid dissemination of various strains of organisms communicable from one individual to another. We know that the rapid passage of an organism from one animal to another tends to increase its virulence, and possibly this aspect is important. In an asylum, gaol, school or factory, the change in the population is small, and where individuals come into close contact with others it is usually with the same people each day. The organisms in such a community are therefore distributed amongst only a relatively small number of individuals. It is probable also that the sleeping accommodation has much to do with the spread of the disease, as the conditions in a military camp give much better opportunities than in a hospital for the insane or in a prison for the passage of organisms from one individual to another.

Is the Incidence of Cerebro-spinal Fever Comparable with that of Pneumonia or with that of Diphtheria?

Diseases like measles, scarlet fever and small-pox are directly and markedly contagious. Practically all persons unprotected by previous attacks, or in the case of small-pox by vaccination, fall victims to the disease on suitable exposure. In diphtheria we know that the disease is directly contagious, but we also know that many persons may, during the course of an epidemic, harbour the organisms without developing the disease. In some of these cases there seems to be an evident reason why these persons do not become ill when the organism present is non-toxic; in certain other cases, however, toxic diphtheria bacilli are present in the throats of "carriers" without producing the disease. In the latter cases the individuals are presumably protected through having manufactured themselves sufficient antitoxin to counteract the toxins produced by the bacilli, which are alone responsible for the disease manifestations. The incidence of cases of cerebro-spinal fever differs markedly from that of any of these diseases.

As regards acute lobar pneumonia, we have strong grounds at present for believing that the pneumococcus is responsible in many cases of this disease. The pneumococcus, however, is considered to be almost universally present at one time or another in the throats of human beings, and the strains isolated from such situations seem indistinguishable from those obtained from cases of acute lobar pneumonia.

The pneumococcus also at times seems responsible for certain types of sore throat, conjunctivitis, meningitis, peritonitis and other conditions.

If the pneumococcus is so universally distributed and so relatively harmless, why does it at times give rise to such severe types of disease as those indicated? Why, further, are certain periods of the year, those more especially associated with certain weather conditions, responsible for definite increases in the number of cases of lobar pneumonia, and why do some individuals show under the same weather conditions a greater liability to the disease? These questions remain at present unanswered, but it seems clear that exhaustive work, accompanied by profuse sweating, followed by exposure to a chill and accompanied by alcoholic indulgence, play some part in producing increases. In other words, we have a universally distributed organism, as a rule non-pathogenic, playing a marked pathogenic rôle under conditions tending to lower the resistance of the individual to infection.

It has not been suggested that in pneumonia we should try to limit the incidence of the disease, which, it may be borne in mind, produces annually in Australia very many more cases and very many more deaths than meningitis has contributed since its first introduction to Australia, by isolating "carriers" of the pneumococcus, so as to prevent spread of the infection. It seems fairly obvious that detection and isolation of such "carriers" would prove useless, on account of the almost universal distribution of the organism, and that such isolation would prove absolutely ineffective in diminishing the incidence of the disease.

Some authorities have estimated that 10% of the ordinary population not knowingly exposed to the disease (cerebro-spinal fever) or brought specially in contact with it are "carriers." If at any period this proportion is really found to be "carriers," it must be assumed that within a period of a year half or more of the population must for a greater or longer period have been "carriers." Unfortunately, data of sufficient amplitude do not seem to exist to show whether a percentage of "carriers" as great as this exists during periods when the disease is non-epidemic. It is probable that, even when the disease is epidemic, the above estimate of 10% is, moreover, too high. The evidence, however, seems quite clear that up to 10% of contacts of cases of the disease may harbour the organism; but it is a matter of common knowledge that cases of the disease amongst "carriers" are uncommon. The danger of association with a case of the disease, as evidenced by the experience of medical men and nurses and of contacts, is evidently not great. As regards ordinary contacts, it is doubtful whether the incidence is appreciably greater than amongst non-contacts. With persons, however, brought persistently in association with cases of the disease there seems to be clear evidence of increased danger, as shown by medical men and nurses occasionally contracting the disease under circumstances which would seem to indicate that, had they not been attending to patients, the disease would not have been contracted. In this disease, therefore, the liability to contract

cerebro-spinal fever from a case of the disease does occur, though this liability is small. In ordinary acute lobar pneumonia there seems to be no reliable evidence of any definite danger in this respect.

To sum up the position of affairs from this resumé, I am inclined to consider that measures taken to detect and isolate "carriers" during an epidemic are little likely to decrease to any recognizable extent the incidence of the disease. On the contrary, however, I am of opinion that, where such detection and isolation of "carriers" can be reasonably accomplished without causing undue hardship or official embarrassment, some little good may result, which, in our present state of imperfect knowledge, may be greater than the data at our disposal would justify us in at present assuming, and that, therefore, such measures may be reasonably carried out.

As regards the introduction of the meningococcus to new countries or fresh localities, the detection and isolation of "carriers" may be manifestly of great importance in stamping out the disease, provided the case or cases of the disease are not manifestations of an infection already widespread.

(To be continued.)

Reviews.

CEREBRO-SPINAL FEVER.

There is a tendency among scientific writers to deal with their subjects without consideration for the known and unknown factors which produce local variations. When the subject-matter of a book is a disease, the climatic influences on both the patients and the causal organisms, the social and economic conditions, the flora, fauna and other environmental peculiarities of the country and modes of procuring, selecting and preparing foods should be taken into account. For these, among other reasons, a treatise descriptive of any disease written by Australian observers should prove of greater value to the profession in the Commonwealth than one composed in other countries. We therefore welcome the appearance of a study of cerebro-spinal fever by two competent Australian observers¹ and applaud the Quarantine Service for having published this work as a service publication. The authors should have expended more care on the literary side of their task, for the defects in composition cannot be overlooked, and the book labours under a serious disadvantage on this account. We hope that a second edition will be required, and that careful editing will remove these shortcomings.

The value of the work lies in the fact that a great deal of the matter is the direct result of personal observations made by the authors during the recent epidemic in Melbourne. In dealing with the ætiology of the disease, they show that only 4.8% of the patients under review had been in direct contact with patients suffering from the disease, and that only 2.3% of the patients came from houses in which another case of the disease occurred. Notwithstanding the large number of cases, the infection spread only to two medical men and eight nurses. Signs of inflammation of the fauces were found in 90% of the patients, although only 40% complained of sore throat. Their investigations in regard to the frequency of carriers of the meningococcus are not complete enough to justify them in making any deductions, and they content themselves by quoting extra-Australian figures. That the coccus gains an entrance in the majority of cases through the naso-pharynx is regarded as practically proved, and the authors enter upon a discussion as to whether the pia-arachnoid is infected directly through the lymphatic channels or whether there is a primary septicæmia, with secondary localization in the meninges. They favour the latter view. The chapter on

¹ Cerebro-Spinal Fever, by N. Hamilton Fairley, M.B., and G. A. Stewart, M.B., B.S., 1916. Quarantine Service Publication, No. 9. Melbourne: Printed by Authority by Albert J. Mullett; Royal Svo., pp. 188.

the bacteriology is short, but suffices for the purposes of the work.

In dealing with the symptomatology, the authors recognize a catarrhal, a septicæmic and a meningeal stage. In about one-half of the patients in the Victorian epidemic there was a definite naso-pharyngitis on admission, and they adduce some evidence in favour of the view that many cases do not progress beyond this. They show that, at the military camp at Seymour, a widespread epidemic of so-called influenza was proved bacteriologically to be a catarrhal form of cerebro-spinal fever. In regard to the septicæmic stage, they accept the evidence as sound that this stage actually exists. Notwithstanding the fact that this stage may be extremely short, or may be masked by the early onset of symptoms of meningeal involvement, it is quite certain that the meningococci must traverse the blood-stream, and the authors are on safe ground in insisting on this point. They call attention to the possibility of the persistence of the septicæmic stage after the meningeal symptoms have become pronounced. In the third or meningeal stage they adopt Sophian's three interacting factors of sepsis, hydrocephalus and meningo-encephalitis. The section on rashes is well given, and that on disc changes interesting and important.

A special chapter is devoted to the behaviour of the blood-pressure in the various stages of the diseases and in the different degrees of illness. The records of the blood and intrathecal pressures are of value and supply food for thought and speculation. The prognostic importance of a low blood-pressure during the early stages, and of a high pressure during the stage of generalized hydrocephalus, is emphasized. Confirmation of the observations of the authors will be required before these statements can be accepted. The theoretical discussion of the causes involved in the lowering or raising of the pressure remain theoretical, and while the arguments employed are ingenious, there are some considerations which might be taken into account which would throw doubt on the conclusions. A discussion of these points in this place would be too lengthy.

A word of praise should be given to the manner in which the chapter on laboratory diagnosis is handled. The chapter on treatment is written with an open mind, and while the authors are confirmed believers in reinforced serum, they have not allowed their imagination to run riot with their facts, as is unfortunately so frequently the case in connexion with the treatment of a disease with a high case mortality. It is doubtful whether the case in favour of anti-serum is a really strong one, and even if the reduction in mortality can be proved to have been due to the use of the serum, its extent is not great enough to justify enthusiasm. The chapter on hexamethylenetetramine appears to us to be like beating the air.

In many ways and from several points of view the work published by the Cambridge University Press of Captain Michael Foster and Captain J. F. Gaskell² may be regarded as one of the best descriptions of cerebro-spinal fever published in the English language. It is worthy of careful perusal, of careful analysis and of careful criticism. The authors have presented their own views, side by side with the views of other observers, and have made an attempt to establish definite teaching on the majority of the problems involved in a proper understanding of the disease. The value of the book lies rather in the chapters dealing with the pathology and bacteriology than in those in which clinical aspects, clinical diagnosis and therapeutics are handled. Some of the opinions expressed in regard to the nature of the infection are unlikely to receive universal acceptance, and have already been challenged by competent investigators.

The authors divide acute cerebro-spinal fever into four forms, presumably for convenience of handling. These are (1) the fulminating, (2) the acute fatal cases, (3) the acute cases with recovery, and (4) the abortive cases. They describe the first type as an illness coming on with startling suddenness and running a uniformly rapid course, terminating in death in twenty-four to thirty-six hours. They regard the condition as one of profound toxæmia, but deny that it is a septicæmia. Such an attitude appears to be untenable, since it is admitted that the point of origin of the infection is the pharynx, that the manifestations of the

condition are a definite acute meningitis and at times a purpuric rash. If an infective process starts as a local infection and spreads either rapidly or slowly to the central nervous system, and especially if a cutaneous eruption manifests itself, it is reasonably certain that the infecting agent has traversed the blood-stream to reach the choroid plexus and the ventricles or spinal theca. It is argued at times that a true septicæmia involves a flooding of the blood with the infective agent; but some organisms may have so much difficulty in establishing a growth in the blood that a very considerable number may be poured into the blood and yet but a few may survive to leave the blood-stream at a point of elective affinity. It is admitted that the meningococcus is a fragile organism, and it would be surprising indeed if it could establish itself rapidly during life in the circulating blood. The presence of a rash means that the organisms themselves, or their disintegration products, are carried by the blood-stream into the skin. Although much in connexion with this disease is problematical, one thing appears to be established, namely, that no soluble toxin is produced. The successful passage of the meningococcus through the circulating blood to the skin and to the central nervous system establishes the point that once this organism ceases to lead a saprophytic existence in the pharynx, a bacteræmia is produced. No doubt the meningitic characters of the infection may overwhelm the bacteræmic or septicæmic manifestations, but this does not justify us in denying the existence of the stage.

The subdivision of the sub-acute and chronic cases into four types appears to us to be just as artificial as the arbitrary division of the acute cases. The authors speak of a suppurative, a recrudescence, a relapsing and a hydrocephalic type. In their account of the pathology of the disease, it appears that the differences of the lesions corresponding to the fulminating and acute and to the sub-acute and hydrocephalic types is one rather of intensity of infection and extent of spread than of any distinctive form of disease. From a clinical point of view, it is doubtful whether all cases could be placed in one or other of these categories. When the authors come to speak of the pathological identity of posterior basic meningitis of infants and certain forms of epidemic meningitis, they arrive on safer ground, and carry scientific conviction by their arguments.

A chapter is devoted to the epidemiology of this affection. The scanty information on which it is based serves to show that, notwithstanding the great amount of work carried out on the bacteriology of this and other infective diseases, our methods of investigation are very meagre, and an entirely new plan of campaign is needed before we can produce evidence of how and why one person falls a victim to the affection and another escapes. All they can state positively is that the disease is caused by the meningococcus, which is passed on from throat to throat, often by persons who show no abnormal signs, either in their pharynxes or constitutionally. They know that the disease is a disease of winter and spring, that it is associated with overcrowding and bad ventilation, and that it may attack infants in a civilian population, and also soldiers in camps. These are facts, but the real epidemiology of cerebro-spinal fever still awaits solution.

We have nothing but praise for the manner in which the authors have presented the bacteriological part of their study. They recognize the work of other bacteriologists and exercise sound judgement in discerning between the doubtful and the acceptable records. In regard to the serological tests, they deal cautiously with the agglutinins, and are apparently inclined to the opinion that the reaction is too variable and not well enough defined to be of practical value. The measurement of the opsonic index is, in their opinion, still less capable of differentiating between the meningococcus and other cocci. They are also satisfied that the complement fixation tests and the precipitin tests are of small value in this connexion. The authors have been wise to discuss these and other suggestions, in order that the reason for their refutation may be placed on record. The plates are excellent, and the general get-up of the book is of the usual high quality which characterizes the publication of the Cambridge University Press. No student of cerebro-spinal meningitis can afford to be without a copy of this book.

² Cerebro-Spinal Fever, by Michael Foster, M.A., M.D., and J. F. Gaskell, M.A., M.D., 1916. Cambridge: University Press; Crown 4to, pp. 222, with 11 coloured and other plates. Price, 15s.

The Medical Journal of Australia.

SATURDAY, DECEMBER 9, 1916.

Flexner's Serum.

It is usually held when considerable difference of opinion exists in regard to the value of a special form of treatment that the claims of the partisans are exaggerated in its favour. Caution is required in arriving at a conclusion when the mortality of the disease to be treated varies under ordinary circumstances. The case for the treatment of epidemic cerebro-spinal meningitis with Flexner's serum has been variously stated since the introducer collected the records of 1,294 cases treated with the serum up to 1913. On the one hand, Flexner claimed that the mortality can be reduced from between 70% and 86% to 30%, and Gardner Robb claimed that the reduction is from 85% to 20%; on the other hand, General Rolleston demonstrated that the mortality of the cases treated with serum in the Royal Navy was actually higher than that of the cases treated by other methods. In the present issue Professor D. A. Welsh puts the case in favour of serum given early and in large doses very strongly, and asserts that this treatment is specific and that the mortality without its aid was 85% and with it was between 12% and 21%. Foster and Gaskell failed to detect a marked benefit from serum, while Fairley and Stewart could only recognize a limited value. In other words, no two observers have obtained the same results from the use of Flexner's serum. From the report of the Special Advisory Committee of the Medical Research Committee (National Health Insurance) upon bacteriological studies of cerebro-spinal fever, we learn that the results of serum have been disappointing to the majority of clinicians. An endeavour has been made to explain the differences in these results. Foster and Gaskell have come to the conclusion after careful consideration that the comparative virulence of the epidemics does not account for the local variations. On the evidence available, it would seem that many of the records cannot be regarded as reliable for statistical purposes, because the number of cases included is too limited. Chance can increase or diminish the mortality per-

centage greatly when but few cases are contained in a series. If all the short series were excluded, we have still very wide differences in the figures. Sufficient evidence is not available to determine whether the lower case mortalities were due to the treatment or to diminishing virulence of the infective agent. Professor Welsh asks us to believe that the unsatisfactory results are wholly due to a wrong method of applying the serum. Netter, and later Sophian, pleaded for large doses of serum to be given early in the course of the infection. Netter's initial dose for an adult was 40 c.cm. In the early days of serum therapy bacteriologists recognized that the volume of the serum containing an antibody was no guide to the clinician. A single cubic centimetre may contain a varying amount of antibody. We are therefore not necessarily dealing with therapeutically large doses when 40 c.cm. of serum are injected. More than that; we can be quite certain that as long as the sera prepared by firms or at laboratories are not standardized according to a uniform formula there must be large variations in dosage between the different batches of serum. It would be logical to speak of intensive treatment if the number of bacteriolytic units introduced were known, but we contend that this term has no meaning when the doses are measured in terms of mass of serum. There is, however, one technical difficulty in this connexion. We have not yet sufficient knowledge to ascertain which form of antibody is active in Flexner's serum. From the available data it would seem as if the bactericidal power were the most important, but certain phenomena suggest that there is something else of equal or possibly greater importance. To measure the therapeutic value of the serum by the agglutinating titre or by the opsonic index does not appear to be permissible. Until there is a more scientific foundation for this form of treatment, clinicians should recognize that all the proposed methods of applying serum in epidemic cerebro-spinal meningitis are arbitrary, although *faute de mieux* they employ this therapeutic agent.

HOSPITALS AND THE PUBLIC.

In another column we publish a record of an interview which chosen members of the Tasmanian Branch of the British Medical Association had with

the Premier of Tasmania, when the position of the Launceston General Hospital was discussed. The matter at issue centred largely around the question whether the Tasmanian hospitals should be open to the whole community or whether admission should be restricted to the needy, who could not command the same treatment from medical practitioners outside the institutions. There are some pertinent facts which must influence those in authority in deciding which policy is the sounder. We take it that no one will dispute that the duty rests with the Government to make adequate provision for the treatment of the poor. Those who have no means of sustenance must be fed at the public expense, and no Ministry can evade this duty in the hope that assistance will be forthcoming from charitably disposed individuals. Similarly, those who have no clothes and no homes must find covering and shelter at the expense of the general public. The same principle applies to the provision of medical attendance of the poor during sickness. Usage has willed it that this duty is not wholly cast on the shoulders of the Government. Where a poor law exists infirmaries and other institutions are supported wholly by the State, and at these institutions the destitute may find an asylum and some medical care. On the other hand, the charitable have zealously guarded their right to contribute wholly or partly to the well-being of the sick poor, because it has been felt that the healing of the sick poor is in reality a work of love. While the charitable public have supplied the financial means for carrying on this work, the medical profession has readily given its services without remuneration in the same cause. In the Commonwealth, two circumstances have modified to some extent the relationship of the public to the hospitals. In the first place, there is but little pauperism, and, consequently, there is less incitement to the public to give freely. In the second place, as the State finds itself relieved of the necessity of instituting poor houses and infirmaries, such as exist in the metropolis of London, it finds itself impelled to make up the deficiencies by subsidies to general hospitals, representing from 25% to 100% of the cost of maintenance. These circumstances, however, do not relieve the State Government of its duty of providing medical care to those persons who cannot afford to

pay for it without assistance. A hospital must remain primarily a place where those whose means are insufficient to meet the heavy expenses often incidental to illness may find maintenance and the services of skilled medical practitioners and nurses. Poverty entitling persons to this form of assistance is relative, but it can be defined without difficulty. Persons whose means are sufficient to enable them to pay for the same treatment outside have no claim whatever on the generous charity of the medical profession.

An attempt has recently been made by Labour politicians to extend this duty of providing hospital accommodation to any citizen who cares to claim it. This innovation is explained by the desire of the politicians to gain in favour with the masses of voters and to secure their positions. If a community is foolish enough to entrust its fortunes to chosen representatives who govern by generosity at other persons' expense and who seek cheap popularity by means of a platform full of catchy planks, it has itself to blame if some of its calculations are a long way out. The proper attitude of the members of the medical profession toward the hospital question is to refuse firmly to attend any well-to-do person at a hospital, while giving their services to the sick poor. The only alternative to this attitude would be to require full fees for attendance and operations and other forms of treatment from the Government, which insists on throwing the doors of its hospitals open to all and sundry. This alternative should not be adopted, unless some guarantee is forthcoming that a sufficient number of beds will be reserved for the poorer members of the community.

SCHOOL CLINICS.

The Minister of Education of New South Wales announced in the Legislative Assembly on November 29, 1916, that a clinic for school children was to be opened in Sydney on Monday, December 4, and that a large number of doctors had already been appointed to attend to the children. This information was given as a reply to the Honourable R. Arthur. The Minister stated that, as far as he knew, it was not a fact that the children were being treated by leading specialists at the Sydney, Royal Prince Alfred, St. Vincent's and other hospitals in the metropolitan area, and added that large numbers of children did not go to the hospitals and never re-

ceived treatment. This matter has been dealt with on many occasions in these columns, when the Minister's predecessor was in office. The members of the medical profession in New South Wales regret that the Honourable A. G. F. James is following the policy inaugurated by the Honourable A. Griffith. On a subsequent occasion the Minister stated that it had been found that 2,051 children required treatment for affections of the eyes, and that 1,426 had failed to obtain it. Similarly, 1,026 children were suffering from ear complaints, and of these 737 had failed to obtain treatment, and of 3,697 children registered as having affections of the nose and throat 2,868 had failed to secure treatment. These figures, we are told, apply to the metropolitan district. There are three matters of importance. In the first place, the Minister stated that the children did not go to hospitals and did not receive treatment. The hospitals are ready to receive these children, and if the Principal Medical Officer would make arrangements for the reception and treatment of children found defective at the inspection, there would be few who would escape treatment. The second point is that over 2,000 children were said to be suffering from ophthalmic defects, over 1,000 from aural defects, and over 3,500 from nasal and faucial defects. The total number of school children inspected in 1915 by nine medical inspectors in the metropolitan district was 35,298, which averages 3,922 children per medical inspector. The result is necessarily extreme hurry over the work, and, consequently, the reliability of the diagnoses made cannot be great. When it is remembered that these gentlemen have to deal with eyes, ears, noses, throats, skin, backs, and other portions of the body, it is manifest that their diagnostic acumen in each special branch of medicine cannot be compared with that of practitioners who have specialized in these departments. We are therefore justified in asking for confirmation of the statement that the number of children suffering from defects of the eyes, ears, noses and throats is as high as has been reported. The third point concerns the announcement that a large number of doctors have already been appointed to the positions referred to in an advertisement which appeared in the public press a short time ago. If these appointments have been made, why have the names of the new medical officers not been published in the *Government Gazette*? Is the Department ashamed of its methods?

THE VAGARIES OF A BOARD.

One of the results of the recent coal strike was the passage into law in the New South Wales Parliament of the Electric Lighting and Gas Emergency Act, 1916, and the consequent appointment of a Board. This Board was empowered to notify the public of Sydney in regard to the conditions under which electric current and gas might be used for the purpose of driving machinery and of lighting. On November 19, 1916, the use of electric current as power was prohibited, except for certain purposes connected with the supply and conservation of food, for water and sewerage services, for use in institutions

for the relief and cure of the sick and for dental work. On November 20 electric current for lighting purposes was prohibited save for public lighting by the City Council and for the necessary lighting of institutions for the relief and cure of the sick. On the following day restrictions in the use of gas for power and industrial purposes, as well as for heating, cooking and lighting were introduced, but it was specifically provided that the prohibition did not apply to the professional work of medical practitioners. Notifications appeared dealing with the hours during which gas might be employed for various purposes at intervals during the continuance of the strike, but no further prohibitions or exemptions were proclaimed in the *Government Gazette*. After a short time, it was announced in the daily press that electric power might be utilized for driving lifts. How and why lifts were exempted from the general rule was not explained. Another exemption was granted to opticians. The Board permitted them to use electric power for the purpose of making spectacles and electric light for the purpose of testing sight. It is to be noted that medical practitioners in Sydney were precluded from using any form of electricity in the exercise of their profession, so that no skiagraph could be taken and no giant magnet could be used, save in a hospital. The prohibition of the use of electricity for diagnostic or therapeutic purposes was sufficiently serious to warrant a protest being made, but this prohibition became intolerable when manufacturers and purveyors of spectacles were exempted in connexion with the testing of sight, which lies outside their province. This action of the Board is all the more reprehensible since the same body recognized the principle of exempting medical practitioners from the rule as applying to gas, in so far as their professional work was concerned. We claim that as long as the supply of coal admits of a limited use of electricity, the public should not be deprived of the boon of modern methods of diagnosis and treatment.

Naval and Military.

The 245th and the 246th lists of casualties were issued on December 2 and December 4, 1916, respectively. The lists contain the names of 315 officers and men killed in action or died from various causes, and of one sister who died of illness. We regret to learn that Captain E. J. Hutchinson is among the wounded. There are no other names of medical men mentioned.

It has been announced that the late Captain Norman W. Broughton, R.A.M.C., was awarded the Distinguished Service Order posthumously for gallantry in the field. On three occasions he helped to dig out men from blown-in dug-outs during very heavy bombardment. Shells were bursting all round him, but he refused to take cover. It was mainly due to his efforts that the majority of the buried men were rescued. It is stated that he had done other fine and gallant work.

We have received requests for a large number of copies of *The Medical Journal of Australia* of October 21, 1916, containing Dr. John F. Wilkinson's article on Starvation and Diet in Diabetes. As this issue is now practically exhausted, we are prepared to reprint this article if a sufficient number of medical practitioners intimate to us before the end of December that they desire a copy. The cost of the reprints will be 1s. each.

Abstracts from Current Medical Literature.

DERMATOLOGY.

(205) Parakeratosis Variegata.

Graham Little records a case of *parakeratosis variegata* of the type described by Juliusberg as *pityriasis lichenoides chronica* (*Proc. Royal Soc. Med.*, June, 1916). The history given by the patient, a girl aged 18 years, was that she began to menstruate when 11, and that the eruption suddenly appeared three months later, with a vivid red rash on the arms and legs. It progressed slowly, and in three years the entire surface was affected, with the exception of the nails. The scalp was scaly, but there was no loss of hair. Epileptic fits occurred during five years preceding the time when Little saw her, and were especially frequent at the time of menstruation. On examination, the eruption was found to be almost universal. There was a fine lichenoid desquamation, with points of accentuated redness as compared with the bluish-red general tone of the skin. The points constituted indefinite maculo-papules. A shadowy effect of network was observed. There was desquamation of the face, which had a blotchy red appearance. The palms and soles were thickened, but the nails were not affected. The skin of the dorsum of certain joints was thickened, presenting some resemblance to *pityriasis rubra pilaris*, but the follicular papules associated with that disease were absent. The mucous membranes were normal. The colour of the eruption varied, sometimes appearing as a vivid, blotchy red, resembling the rash of measles. At its inception the eruption was mistaken for a measles rash. For five years there had been but little change in the character of the rash, except for the variation in colour. There had not been any pruritus. The thoracic and abdominal viscera were normal. There were some enlarged glands in the neck. Ammoniated mercury ointment (grs. x, ad 3 i.) had been tried for two years, and, like all other forms of treatment previously employed, had not effected any improvement. The case resembles an affection described by Fox and MacLeod as *parakeratosis variegata* and classified by them with *pityriasis lichenoides chronica* of Juliusberg. The analogy between the present case and members of this group is seen in the youth of the patient, in the character of the eruption, with its resemblance to a receding lichen planus and its wide distribution. The absence of constitutional symptoms, and its resistance to treatment, are also qualities common to the two affections. In one case only of the series described by Fox and MacLeod was the onset acute.

(206) Purpura Due to the Meningococcus.

A case of meningococcal meningitis with unusual purpuric manifestations is reported by Charles Sharpe (*Journ.*

Cutan. Dis., September, 1916). The patient was a child, 3½ years of age. He had been sent to hospital with a diagnosis of measles and purpura. On physical examination, the most notable feature seen was a widespread purpuric eruption, the larger lesions of which had a remarkable vermilion-coloured border. The lesions varied from small petechiae to suffusions 4½ inches in diameter. The face, trunk and limbs were extensively affected, while the scalp escaped to a great extent. There were conjunctival hemorrhages, and the eyelids were also involved. Petechiae were present on the buccal mucous membrane on the hard palate and on the tongue. A blood culture was taken and a Gram-negative diplococcus was isolated. This organism was successfully inoculated on glucose acetic agar tube. The patient died three days after admission. Immediately after death sections of the skin were obtained from the vermilion border of the purpuric lesions. Gram-negative cocci were found in the sections. The organisms were more readily recognized after staining with Pappenheim-Saathoff's stain than by Gram's method. They were situated both intra- and extra-cellularly, and were seen within the capillaries, as well as free in the tissues. The author states that the meningococcus has been found in the secretions of the nose, eyes and bronchi in pneumonia, in the pus from joint affections, in the urine, in subcutaneous abscesses and in the blood, but that he has been unable to find the records of a case in which it has been demonstrated in the skin. He calls attention to the fact that when spinal symptoms are marked the cutaneous symptoms are often negative and *vice versa*.

(207) Blood Counts in Parasitic Skin Diseases.

Albert Strickler (*Journ. Cutan. Dis.*, October, 1916) points out that the study of the blood condition in skin diseases has been general rather than limited to one affection. In a previous communication he has shown that the majority of cases of scabies are associated with a distinct eosinophilia. He has undertaken a study of the blood picture in a series of cases of ringworm, and has compared his results with those obtained in *pediculosis corporis*. In addition to his own cases, he has taken into account the records of nine previously published cases. He differentiates the cases treated locally from those treated with vaccine. It appears that the average count carried out in 60 cases revealed 37.4% of small lymphocytes. In 20% of his own patients there was no increase in the number of these cells. There was a slightly greater increase in those patients treated by vaccine. He is of opinion that this increase in the number of small lymphocytes may be of value in the differentiation between ringworm of the scalp and other skin affections. *Pediculosis corporis* does not produce any change in the blood picture. He has also studied the blood from four cases of favus, and states

that there seems to be an increase in the number of the small lymphocytes in this disease as well.

(208) Radio-Sensibility.

C. A. Simpson discusses the sensitizing effect of cold on surfaces treated by X-rays (*Americ. Journ. Roentgen.*, October, 1916). The age of the patient, the area of the body exposed to the influence of the rays and local anæmia induced by injections of adrenalin are well-recognized factors contributing to an increased susceptibility of the skin. The moisture of the surface, the reaction of the sweat and the condition of the vaso-motor nerves of the skin are also said to influence this susceptibility. Heidingsfeld has described a radio-dermatitis of the second degree following three mild exposures in a case of dermatitis hiemalis. The aetiology of this condition and the fact that the skin is dry and free from sebaceous secretion give rise to the suggestion that the sensitization is derived from the effect of cold on the skin. The author has experimented on 38 cases of exophthalmic goitre, which he treated with massive doses of X-rays. When the patient was placed at rest and ice was applied to the neck a temporary improvement resulted from the radiation. He experienced four severe skin reactions after 3½ Hampson units filtered through 1 mm. aluminium and five layers of chamois leather. In some cases the surface of skin to which ice had been applied reacted to the rays while the surrounding skin remained normal. He points out that he regards the ice-bag as so powerful a skin sensitizer that he allows only those patients to use it who have derived marked benefit from its use in the past. In these cases he never exceeds 3½ Hampson units.

(209) Dermatitis Due to the Secretion of a Beetle.

P. H. Ross records the case of a medical man who was in bed in hospital in Nairobi, East African Protectorate, when a beetle, which was identified as *Paederus crebripunctatus*, Epp., hit the wall above his head and fell on to his forehead (*Journ. Tropical Med. and Hyg.*, September 1, 1916). The beetle walked across his forehead and apparently left an irritating secretion behind. On the following day a bright vesicular streak, similar to the effect of strong acid on the skin, was noted. Unless the beetle is irritated, it does not secrete the fluid. The author has evidence that the beetles may rest on the hand or wrist without causing any ill-effects. One man rubbed a beetle hard on to his forearm and suffered the penalty.

BIOLOGICAL CHEMISTRY.

(210) Regional Lipolytic Activity.

C. Quinan (*Journ. Med. Research*, September, 1916) points out that all tissues are able to accelerate the cleavage of ethyl butyrate. The dynamic values obtained by introducing equal weights of the tissues into systems containing

ethyl butyrate and by estimating the acid formed in twenty-hours are characteristic for each tissue, and are proportional to the cellularity of the respective structures. Researches upon endocellular lipase derive their validity from the supposition that a fat-splitting ferment is present in different parts of the body. The ferments can be dissolved by appropriate solvents. The quantity present in a given weight of tissue can be estimated by measuring the formation of free acid under similar conditions. It appears possible that the degree of endocellular lipoclastic activity expresses a definite function of protoplasm. A factor can be determined for each tissue in terms of ester cleavage by a unit of tissue in unit of time. In normal tissues within certain limits this factor varies relatively to the amount of protein in the weighed unit of tissue. In conditions of intoxication with disturbance of nutrition the dynamic factor becomes less than normal and fat becomes visible microscopically within the cells of the tissues. In this connexion it is useful to discriminate between the reaction occurring in the water-soluble phase of fat in the cytoplasm of the cell and the hydrolytic cleavage of neutral fat by the lipase of pancreatic juice. In the human brain both white and gray matters accelerate the splitting of ethyl butyrate. Each main histological subdivision has a constant dynamic factor. All regions of the cerebral cortex have approximately the same factor. The caudate nucleus and the optic thalamus have the same factor, which is slightly higher than that of the cerebral cortex. The gray matter of the cerebellum has the factor with the highest value of any of the structures of the brain. The white matter of the corpus callosum and the centrum semiovale are alike in lipolytic activity, though much less active than any other part of the brain. These results suggest that these differences depend upon differences in the structural elements of the tissues.

(211) Purification of Water.

T. B. Shaw describes the gradual adoption of chlorine and of disinfectants yielding chlorine for the sterilization of drinking-waters (*Journ. Royal Naval Med. Service*, October, 1916). For many years boiling with or without filtration has been the chief method of sterilizing water in the Naval Service. This mode of purification is, however, cumbersome. Chemical processes have been tried for many years. One of the methods first employed was the addition of small quantities of bromine. Permanganate of potash has been used, and also bisulphate of soda. To be useful for chemical purification some substance is needed which can be employed in small amount, and which does not interfere with the potability of the water. The use of preparations liberating chlorine has gradually replaced all other methods of destroying pathogenic micro-organisms in drinking-water for military purposes. Bleaching powder and chlorinated soda have had a wide vogue for some years.

Improved processes for making liquid chlorine have led to the introduction of chlorine in cylinders for rapid sterilization. A recent order by the Admiralty enforces the use of chloros. This is a liquid preparation of sodium hypochlorite containing one-seventh of its weight of available chlorine. The preparation is stable if kept undiluted in a dark place. One-third ounce is added to each 500 gallons of water, or one and a half ounces to ten tons of water. This is equivalent to an addition of one part of chlorine to two million parts of water.

(212) Injection of Hæmoglobin into Men.

A. W. Sellards and G. R. Minot have studied the effects of injecting hæmoglobin into men suffering from different conditions of anæmia (*Journ. Med. Research*, July, 1916). Human blood has been used as the source of the hæmoglobin. The blood has been spun for a brief period in the centrifuge, to deposit the corpuscles but to leave the platelets in suspension. The serum plus the "leucocytic cream" on the surface of the layer of erythrocytes has been removed with a pipette, and the remaining red cells washed once with saline solution. The corpuscles were spun on this occasion for a sufficient length of time to pack them firmly. The red corpuscles have been laked by the addition of four volumes of distilled water warmed to body temperature. The solution of hæmoglobin has been made isotonic by the addition of a concentrated salt solution. The solution becomes turbid and, on microscopic examination, the ghosts of the laked cells are clearly visible. These remnants of the erythrocytes have been removed by the centrifuge. A clear red fluid results, which no longer becomes turbid on the addition of strong salt solution. To avoid any need for sterilization the corpuscles have been treated aseptically from the beginning. The solution has been kept on ice and used within twenty-four hours. In the examination of urine the specimens have been examined with the spectroscope and the highest dilution determined at which the two bands of oxy-hæmoglobin could be detected in a layer one centimetre deep. The injections have been made at first intramuscularly, and, when no ill-effects have occurred, the subsequent injections have been given intravenously. In sixteen normal persons amounts of hæmoglobin up to that obtained from 25 c.cm. red corpuscles have been injected intravenously without any excretion of hæmoglobin. The hæmoglobin from 28 c.cm. of red cells caused slight hæmoglobinuria, and that from 33 c.cm. intense hæmoglobinuria. Injections made with similar quantities in cases of pernicious anæmia and secondary anæmia gave rise to no excretion of hæmoglobin. The administration of the hæmoglobin from 13 c.cm. erythrocytes to a patient suffering from hæmolytic jaundice caused a very intense hæmoglobinuria. A moderate degree of hæmoglobinuria has

been produced by the hæmoglobin from 15 c.cm. corpuscles in a patient suffering from infectious biliary cirrhosis and by the hæmoglobin from 25 c.cm. in a person the subject of myelogenous leukaemia. The authors conclude that distinctly less hæmoglobin is needed to produce hæmoglobinuria in patients in whom increased destruction of red corpuscles is taking place. They believe that they have demonstrated that the injection of hæmoglobin offers a safe and satisfactory means of studying the metabolism of blood pigments in man.

(213) Uric Acid in the Tissues.

H. G. Wells has isolated relatively large amounts of uric acid from the tissues of a girl who had practically complete suppression of urine for nine days as the result of taking 17 grains of perchloride of mercury (*Journ. Biological Chemistry*, September, 1916). The patient had been previously in good health. She lived nine days, during which she excreted only 96 c.cm. of urine, despite all attempts to incite renal activity. During this period she was practically fasting, and such food as was administered was of a purin-free nature. The organs were removed one hour after death and kept on ice before examination. Four separate lots of tissues were subjected to chemical examination. These lots consisted of (1) 1,750 c.cm. blood, (2) 1,350 gm. liver, (3) 1,100 gm. intestines and stomach washed free from their contents, and (4) 1,650 gm. mixed viscera made up of lungs, spleen, kidney, uterus and ovaries and 250 gm. muscles. From the blood there was recovered 25 mg. crystalline uric acid, from the mixed viscera 166 mg., while no uric acid could be isolated from the stomach, intestines and liver. As the uric acid was separated in a pure form the amounts recovered represent minimal amounts, since the losses in the purification are neglected. As a control, organs were taken from four other patients and treated in the same way. No uric acid was recovered, except 26 mg. from the liver of a patient dead from croupous pneumonia. It is therefore certain that an accumulation of uric acid occurs in the body after complete suppression of urine. These results suggest that the idea that the tissues of the human body destroy uric acid is not well founded.

(214) Artificial Diets Containing Lactose.

J. C. Drummond has studied the growth of rats on artificial diets (*Biochemical Journal*, March, 1916). He finds that the rats do not grow at all on a diet of purified proteins carbohydrates, fats and salts. Before growth can occur, both fat-soluble and water-soluble accessory substances must be added to the diet. Lactose prepared from milk contains a substance which may act as a growth-promoting accessory substance. The substance is soluble in water and in alcohol. It is not destroyed by heating it for six hours to the boiling-point of water.

British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch was held on October 27, 1916, at the B.M.A. Building, 30-34 Elizabeth-street, Sydney. Dr. Sinclair Gillies, the President, in the chair.

Professor D. A. Welsh opened a discussion on the prophylaxis and treatment of cerebro-spinal meningitis. He claimed that a *prima facie* case had been made out in favour of using anti-serum in the treatment of epidemic meningitis. In dealing with mild cases he expressed his view to the effect that, since it could be proved that anti-serum was of value, it should be used in all cases as early as possible. Some observers have been discouraged because unfavourable accounts of the treatment have been published. He considered that this was an untenable position, and he trusted that members would not agree with it. In his opinion the unfavourable results were more often due to the fact that sufficient quantities of serum had not been introduced early enough than that the anti-serum was unsuitable.

The speaker then proceeded to analyse the cases in which the intensive treatment had been employed. In order to arrive at an accurate determination of the value of the treatment he referred to four series of cases. The first series consisted of 20 serious unselected cases at the Royal Prince Alfred Hospital and 14 military cases in the camps, in which no treatment, or too little treatment with anti-serum had been given. The case mortality in this series of 34 cases was 85%. The deaths numbered 29. The second series comprised a few experimental cases, with which the speaker had dealt in his previous communication to the Branch (see *The Medical Journal of Australia*, August 12, 1916, p. 113 and p. 124). The third series consisted of military and civil cases observed by Dr. Wolfe Brown. All the 33 cases of this series were serious ones. There were four deaths, which yielded a case mortality of 12%. Some of these cases, however, were treated at the beginning of the crusade, before the full significance of the intensive treatment had been recognized. He held the opinion that three of the four deaths occurred in patients who were not treated as energetically as he would now advise. The fourth series consisted of cases to be described by Dr. Nowland. The patients numbered 35. They were treated in the Royal Prince Alfred Hospital. There were 11 deaths. Two of the deaths occurred immediately after or during parturition in the course of the meningeal infection, and should therefore not be regarded as direct failures of specific treatment. Death was due in the first case to puerperal sepsis, and in the second to eclampsia. Of the nine remaining, two patients died with well-marked broncho-pneumonia. He was disinclined to consider these cases as instances of actual failure of specific treatment, but rather as instances in which the anti-serum had not had a chance of success. In four the condition advanced to a stage when the spinal infection had disappeared, fluid was not obtainable by lumbar puncture, and cerebral pressure symptoms had increased. The post-mortem appearances were those of acute hydrocephalus, with greatly dilated ventricles, but no free fluid over the brain or cord. The intensive treatment in these cases had been applied on the third, fourth and fifth days of disease, and although he regarded this as much too late, he considered that they should be entered as failure of the anti-serum treatment. When specific treatment was delayed, complications were liable to arise which were outside the curative effect of the serum. He referred to broncho-pneumonia and mechanical complications, such as hydrocephalus. The last three deaths occurred in cases of uncomplicated meningitis, which he admitted to be failures as far as the serum treatment was concerned. Excluding the deaths from broncho-pneumonia, there were seven fatal cases. This represented about 21% case mortality.

He noted a considerable difference between the mortality in Dr. Brown's series and that in Dr. Nowland's series. The majority of the patients under Dr. Brown's care were in the military camps. These patients reported early, and treatment therefore had a better chance than it had in Dr.

Nowland's series at the Royal Prince Alfred Hospital, where the majority of the patients applied for treatment late. In one instance the patient had been unconscious for three days before admission to the Royal Prince Alfred Hospital. He also cited the case of two sisters who had been ill for three and five days respectively before admission. The one recovered, while her sister, who had been ill two days longer, did not respond to the treatment and died of broncho-pneumonia. In considering the results obtained in these two series of cases, Professor Welsh arrived at the conclusion that the relative failure in Dr. Nowland's series was due to the greater delay in commencing the specific treatment, and was therefore one of the strongest arguments in favour of proper intensive methods.

He reiterated that the treatment must be intensive and immediate. He was satisfied that the antiserum should be introduced intrathecally. On the other hand, it seemed to him that some form of treatment should be applied to the blood. Arguing by analogy with influenza, he conceived the opinion that the meningococcus reached the central nervous system only by accident, as it were. He therefore suggested that antiserum should be given in addition, either by intravenous or by subcutaneous injection. He preferred the subcutaneous route, as it appeared to him to involve less risk. The antibodies got into the blood-stream eventually. In conclusion, Professor Welsh spoke of the advisability of employing polyvalent meningococcus vaccines.

Dr. Wolfe S. Brown read a paper entitled "Some Observations on Cerebro-spinal Fever in the Military Camps and Country Hospitals in New South Wales." The text of the paper will be found on page 487 of this issue.

Dr. R. E. Nowland read a paper on the treatment of cerebro-spinal meningitis (see page 489).

Dr. J. B. Cleland read a paper by Dr. W. G. Armstrong on the administrative and epidemiological aspects of cerebro-spinal meningitis (see page 492).

Dr. J. B. Cleland also read a paper by himself on some aspects of the aetiological side of cerebro-spinal meningitis (see page 496).

Dr. W. Keith Inglis also read the notes of a case of suppurative meningitis (see *The Medical Journal of Australia*, December 2, 1916, p. 474).

Dr. W. F. Litchfield referred to Professor Welsh's communication to the Branch in August, and stated that it had aroused considerable interest. He held that the post mortem examination conducted in one of Professor Welsh's cases was of much importance. Many observers had been present, as it was felt that the appearances after death might throw some light on the action of the treatment. He had had several cases of cerebro-spinal fever at the Children's Hospital. Since August of the present year he had had five cases, and before that there had been three under his care. All these eight patients had recovered. He was of opinion that he had been very fortunate; but the fact remained that there had been a series of eight cases without a death. Since July the total number of cases at the hospital had been 17. Of these, 14 had been treated with serum. The total number of deaths was 7, while five of the deaths had occurred among those treated with serum. The total mortality was therefore 41%, while the mortality of the serum-treated cases was 35.7%. These cases could not be compared with Dr. Brown's cases, because the majority of the patients had not come into the hospital for treatment for several days after the onset. The interval before treatment was applied had reached two weeks in one case. They had not given children the quantity of serum that Dr. Brown had spoken of. The usual dose given was 15 c.c.m., which was repeated once or twice after intervals of 24 hours. Five of the patients were under two years of age, and the great majority were under three. In some of the cases there had been difficulty in regard to the diagnosis. One child had been admitted with a diagnosis of pyloric stenosis. After it had been in the hospital for some days, stiffness of the neck developed. Lumbar puncture was carried out and some turbid fluid collected, from which the meningococcus was isolated. The child recovered. The age of this patient was five months. Another child had been treated for enteric fever for two weeks before the diagnosis of meningitis was established. A third child had been ill for eight weeks when the symptoms of cerebro-spinal fever developed. It died within a few hours.

In the next place, Dr. Litchfield spoke of the value of morphine in the treatment of this condition. He referred to a female patient, aged 47 years, to whom serum had been given. There was much pain. One-sixth of a grain of morphine had been given every six hours for three days with benefit. The patient recovered in about eight days.

The question of "dry tapping" had been referred to. He had experienced in the case of a child a striking instance. His resident medical officer had punctured and had failed to evacuate any fluid. He himself had tried again and again, but always with the same result. One day later puncture was done again and between 40 and 50 c.cm. of turbid fluid had been evacuated. Serum had been given in this case and recovery ensued.

Dr. Litchfield asked for more information in regard to the chronic cases. Perhaps Professor Welsh could suggest why it was that the meningitis in these cases did not clear up in spite of enormous quantities of serum. Was it because the particular strain which persisted in these cases was not killed by the specific serum employed?

Dr. A. E. Mills, in thanking the readers of the various papers, stated that he had been struck by their remarkable optimism. It was a cheery sign. But he was a little doubtful whether this optimism was quite justified. He raised the question whether it was permissible on the part of Professor Welsh to exclude the broncho-pneumonia cases from his series in arriving at the mortality. After all, the pneumonia had been caused by the meningococcus, and the patients had died in spite of the intensive treatment. He would add these cases, and the mortality rate would consequently be raised. He was not prepared to say anything against the intensive treatment.

Turning his attention to the serum itself, he found himself in difficulty. There did not appear to be any standardization. In Melbourne they used their own serum. When they employed imported serum the results had been bad. The Melbourne serum had yielded much better results. He held the opinion that, until a great number of cases had been collected and the conditions carefully analysed, it was dangerous to form any definite conclusions. By doing so they would render themselves liable to mistakes. He would be a strong advocate of serum treatment if it could be shown that the serum had an antitoxic value. He thought that it was advisable to give intravenous injections of serum in the intervals between the intrathecal injections. It was quite uncertain how the serum spread up the spinal canal.

Dr. Mills had been especially interested in Dr. Inglis's case. It sounded remarkably like a case of anaphylaxis. The only flaw in this interpretation was that the death was somewhat delayed. Intense dyspnoea and oedema of the lungs were symptoms of anaphylaxis.

Referring to Dr. Brown's dictum in regard to clear serum, he could not accept the view that turbidity was a necessary accompaniment of meningococcal infection. He held that the spinal fluid should be examined in the laboratory in every case. He pointed out that the source of the fluid was the choroid plexus. The choroid plexus contained vaso-motor nerves, notwithstanding the statement to the contrary of some physiologists. He referred to some observations made by Professor Wilson which had satisfied him on this score. It seemed to him extraordinary that the vessels should have muscular fibres if they did not have the power of contraction. He suggested that in certain cases toxins in the blood acted on the vaso-motor nerves in the choroid plexus. If they produced a contraction of the vessels there would be no outpouring of fluid, while, when the vessels were dilated, the fluid would increase in quantity. He cited a case of a boy in whom there were definite signs suggestive of pneumonia. The child was drowsy and the temperature was raised. There were, however, no signs in the lungs. Lumbar puncture revealed 30 or 40 c.cm. of clear fluid. Bacteriological and chemical examination showed that it was normal. On the following day a patch of pneumonic consolidation appeared. The patient recovered. Another boy injured his thigh and got a septic infection. The wound in the thigh was caused by a splinter of wood. The splinter was removed and the wound was healing. When admitted into hospital he was suffering from generalized rheumatic pains and general stiffness of the body, head and neck. Between 40 and 50 c.cm. of clear fluid was ob-

tained by lumbar puncture. It was devoid of organisms and of cellular elements. The symptoms lessened, and the boy recovered within a few days. In these cases he held that the toxins circulating had increased the outflowing of fluid through the vessels of the choroid plexus. In Dr. Inglis's case the infecting organisms also produced a toxin, which incited the vaso-motor nerves to dilate the vessels in the choroid plexus.

The speaker considered that an increase of cellular elements in the fluid would probably not appear at the very beginning of the infection to such an extent as to produce turbidity. He held strongly that a macroscopical examination was not sufficient. A full microscopical examination should be carried out. It was often unnecessary to wait for the results of a bacteriological investigation. The cytology of the fluid frequently gave valuable information. Returning to his suggestion concerning the action of toxins on the vaso-motor nerves, he ventured to explain "dry tap" by assuming that the toxin in these cases prevented the excretion of fluid. Later, as the vessels became dilated the fluid poured out.

Dr. Mills advocated the use of autogenous vaccines. He expressed strong doubts as to the value of urotropine. A minute trace sometimes found of formaldehyde was unlikely to have any action on the growth of the meningococcus.

Dr. Sinclair Gillies expressed the opinion that Professor Welsh had presented a stronger case in favour of anti-serum with his more recent series than with his first series. He would like to know whether Dr. Brown had treated all his cases himself. He also asked whether there had been a bacteriological confirmation in every case. If this were not the case, the evidence would lose considerably in value. Was Dr. Inglis's case included? He was inclined to agree with Dr. Mills that Professor Welsh was an enthusiast. He thought it probable that Dr. Inglis's case was one of anaphylaxis. The lesions were those of a vertical purulent fibrinous lymph exudate; why should it not have been a pneumococcal meningitis? He held that it was incorrect to assume that a turbid fluid was always diagnostic of cerebro-spinal fever. It occurred in pneumococcal meningitis. In spite of the various opinions expressed, he adhered to his opinion that it was worth while giving urotropine in meningitis.

In discussing the mortality figures which had been presented, he pointed out that different epidemics were mild or severe, and that a very much larger series of cases was needed before a definite claim could be made out that the serum was a specific. In the meantime he thought that Professor Welsh was justified in recommending that serum should be given in every case.

Professor Welsh, in his reply, advocated the abstraction of as much fluid as could be obtained, in order that a larger quantity of serum might be induced to enter the lateral ventricles. The differences in the response to the intensive treatment were, in his opinion, due to the time when it was applied. If the anti-serum had been given early the response was prompt; while, if the treatment were delayed and intractable complications were encouraged to develop the response was much less marked. He did not agree with Dr. Cleland that there was no septicæmic stage in cerebro-spinal meningitis. He was prepared to admit that Dr. Inglis's case might have been an instance of anaphylaxis. On the other hand, an acute pulmonary oedema after other inhalation is a recognized, though rare, complication.

He held that Dr. Litchfield's statistics supported his case in favour of the intensive treatment. Dr. Litchfield had recognized the necessity of giving the serum in large doses and in an early stage. Turning to Dr. Mills, he repudiated the suggestion that he was an optimist, unless he could get the cases early. He explained that it was not his intention to exclude the broncho-pneumonia cases from the series, but that he was attempting to estimate the statistical value of complications which the anti-serum was powerless to avert, and to treat.¹ He stated that means

¹ In his statement of the case mortality in Dr. Nowland's series, Professor Welsh started with 11 deaths among 35 patients. Two of these were excluded because the death was associated with a puerperal condition. Of the remaining nine deaths, the two broncho-pneumonia cases were deducted, leaving seven deaths. The mortality rates would be 31.4% for the whole series, 27.3% for the 33 cases, with nine deaths, and 22.6% for the 31 cases, with seven deaths.—Ed.

existed for standardizing the serum by the measurement of the agglutinating and opsonizing activity, and also in regard to bactericidal power. A high bactericidal value was found when it was specific for the organisms met with. The experience of Dr. Brown and Dr. Nowland had been that Parke Davis & Company's serum was clinically more effective than the Melbourne serum. In regard to urotropine, it could be shown that very minute traces of any antiseptic inhibited the growth of this most sensitive organism.

Dr. Brown stated that he had been present when all the patients had been treated; practically all the cases had been confirmed bacteriologically. Dr. Inglis's case had not been included.

Dr. Cleland maintained that a true septicæmia involved a condition in which the organism was multiplying appreciably in the blood.

Dr. Inglis considered that his case was one of meningococcal infection, but even if it had been pneumococcal the fact remained that fluid obtained by lumbar puncture appeared to be quite clear, although examination after death a few hours later had revealed pus in the sulci over the vertex of the brain. He referred to a case in which the pus was distributed in a similar manner. In this case the cerebro-spinal fluid contained a Gram-positive coccus, which was probably the pneumococcus.

MEDICO-POLITICAL.

A special General Meeting of the Tasmanian Branch was held in Hobart on November 26, 1916. Dr. G. E. Clemons, the President, in the chair. Letters were read from many members who were unable to attend the meeting, but who wished to record their strong disapproval of the admission of well-to-do patients into State-aided hospitals.

It was resolved unanimously:—

That this meeting disapproves of the Premier's statement that people in well-to-do circumstances should be treated in State-aided hospitals as well as the poor.

It was also resolved:—

That if the Premier persists in his policy of permitting admission of well-to-do patients into State-aided hospitals, the honorary system must of necessity cease.

The Honorary Secretary was instructed to communicate the contents of the resolutions to the Premier, and to ask him to inform the Council what his intentions in this connexion were.

Dr. George H. Gibson and Dr. Stuart Gibson were elected members of the Branch.

THE LAUNCESTON GENERAL HOSPITAL.

The State Premier of Tasmania, the Honourable W. H. Lee, received a deputation of the members of the Tasmanian Branch of the British Medical Association on November 15, 1916. The Branch was represented by Dr. G. E. Clemons (President), Dr. G. H. Hogg, Dr. G. Sprott, Dr. A. H. Clarke, Dr. T. H. Goddard, Dr. T. C. Butler, Dr. R. G. Scott, Dr. E. W. J. Ireland, Dr. A. E. Hayward (Honorary Secretary), Dr. D. H. E. Liles and Dr. C. M. Atkins.

In introducing the deputation, Dr. A. H. Clarke pointed out that the members of the Tasmanian Branch had noticed with much concern that persons in good circumstances were being admitted to an institution that was in receipt of State subsidy, and were obtaining treatment. This practice meant competing with private institutions which received no State subsidy. They maintained that an institution supported by the State was intended for people of small means who were not able to pay a doctor's fees or the charges of a nursing institution for ordinary treatment and who required treatment at a reduced rate.

Dr. Clemons reminded the Premier that a few months previously the Council of the Tasmanian Branch had forwarded a resolution of protest on this subject to the Premier. The Premier had replied in the following terms: "I have placed myself in communication with the Board of the Launceston General Hospital, and they advise me that the statement 'that a large number of well-to-do patients have been admitted' is not correct. For the past 25 years nearly all applicants for medical or surgical treatment have been

admitted, and for such treatment have been charged according to their ability to pay, after full investigation having been made by the Board and the Visiting and Finance Committees. With regard to the statement that the practice of admitting well-to-do persons is contrary to the use of a Government general hospital, and seriously affects the interests of the medical profession, it is pointed out that the practice at the Launceston General Hospital is in conformity with that of all the general hospitals throughout Australia, as far as is known to the members of the Board. However, the Board considers that the financial position of any person seeking for admission for treatment should not be the standard by which his admission or rejection is decided. The hospital is a Government institution and maintained almost exclusively by the taxpayers, and it is contended by the Board that any person, whether poor or well-to-do, has a right to the benefits of the institution. The Board also emphatically denies that any abuse exists in this regard, and that the proof that such does exist rests with those making the accusation." Dr. Clemons categorically answered the points in this letter. He quoted from the report of the Royal Commission held in 1905, to show that there had been abuse by the admission of well-to-do persons to the hospital.

Dr. Elkington, the Chairman of the Commission, had reiterated this statement five years later. In regard to the contention that the practice in Launceston was in conformity with that in all general hospitals throughout Australia, Dr. Clemons read letters received by the Honorary Secretary from the Honorary Secretary of the New South Wales Branch and from the Secretary of the Victorian Branch of the British Medical Association. The Honorary Secretary of the New South Wales Branch had written that, except in emergencies, he knew of no general hospital in New South Wales where admissibility was not determined by financial position. The Secretary of the Victorian Branch had replied to the effect that the same matter had been before the Council of his Branch recently in regard to three hospitals in Victoria. Representations had been made to the Inspector of Charities, who was sympathetic with the views of the medical profession, and was making enquiries concerning the extent of the abuse, with a view to rectification.

Continuing, Dr. Clemons stated that every time a well-to-do patient was admitted to the wards of the general hospitals in Launceston, Hobart, or other cities, he not only exacted more attention from the nurses and doctors because of the fees paid, but actually excluded some poor person who ought to be in his place. He could hand the Premier a list of names of people who had no right to be in a general hospital. There was no doubt that the practice of admitting rich and poor alike was the policy of the Launceston Hospital Board, and the deputation would like the Premier to tell them whether it was also his policy.

Dr. Hogg stated that the Chairman of the Launceston Hospital Board had admitted that ten rich men had been treated in the hospital during last year. That in itself was proof of abuse.

The Premier interposed and remarked that he understood that four of the ten had been sent to the hospital by medical men. He raised the question whether these well-to-do patients were not repeatedly sent in by medical practitioners. Dr. Hogg replied that he knew of no practitioners in Launceston who sent rich patients into the hospital, and Dr. Sprott contended that, even were such action taken, it would not justify the hospital authorities in admitting them.

Dr. Hogg contended that if the Government permitted the abuse to continue at Launceston they would practically be adopting a policy of the nationalization of hospitals, and would be admitting a policy in favour of nationalization of the medical profession.

Dr. Gregory Sprott maintained that if the Government adopted the principle that a general hospital should be opened to rich as well as to poor, the hospitals would have to be nationalized, and the Government would have no right to ask members of the medical profession to take any part in hospital work in an honorary capacity. If the Government endorsed this principle, the Tasmanian Branch of the British Medical Association, which comprised about 90% of the members of the profession, would be compelled to re-

consider its position and to take such steps as were considered necessary.

Dr. Scott said that the members of the profession in the south were in full sympathy with their colleagues in the north. They would not continue their hospital work as honorary medical officers if wealthy people, who should be treated outside, were admitted into the hospital.

The Premier, in his reply, said that he regretted that there should be friction between the hospital in the north and the local medical men, because he thought it desirable, under the existing conditions in which they utilized the services of medical men in an honorary capacity, that the best relations should exist between the hospital and the medical men. Unfortunately, there had been friction for some considerable time, particularly in Launceston, over this matter. Charges had been made, but he would not say whether they were with or without foundation. He thought that in the majority of cases the trouble had occurred some years ago, during the term of office of the late Medical Superintendent, Dr. Ramsay. There appeared to have been considerable friction, some people had called it jealousy, but he would not use that term, because of the fact that, rightly or wrongly, a great many people placed considerable value on the services of that gentleman, and had strained a point in their endeavour to get under his care in the hospital. Under such circumstances, he could quite readily understand the possibility of abuse creeping in and of the Board becoming a little bit lax in carrying out the administration of their duties, but he had been given to understand that of late, at any rate, many of the abuses mentioned by the members of the deputation had been remedied.

Dr. Hogg stated that Dr. Parker had resigned his appointment last year because he had found that he could not secure the operating-room to operate in.

The Premier said that this was a statement on one side of the case. He had not the version of the Hospital Board before him. He did not doubt Dr. Hogg's statement, but he wished to hear the other side before he could arrive at any conclusion. Since the majority of the abuses had been remedied, the matter was narrowed down to a question of policy in regard to well-to-do patients. It was a difficult problem to deal with. He was not quite prepared to take up the position that hospitals should be confined absolutely to poor and indigent persons. If that were so, it would become a question of where they were going to draw the line, and whether any fees should be charged at all. That was the only logical sequel to the Board's contention. If this proposal were pressed, the Government would have to consider it from the point of view of expense. If they made all hospitals for the treatment of the poor only and charged no fees at all there would be a considerable drain on the finances of the State, much heavier than at the present time. In this connexion they had also to remember the straitened times in which they were living. It seemed to him to come down to a question of discretion on the part of the members of the boards of management in controlling these hospitals. In the majority of cases the boards were not careful enough in regard to getting the fees in. He would not commit himself definitely as to whether the rich should not be treated but it appeared to him that the hospitals were set up for the benefit of the general public, and if a man considered that he could get equal or even better treatment at a general hospital and the advantage of nursing attention which he would probably not get at home, it would become a serious matter to say to any citizen of the State: "We will not allow you to do it, even provided you pay on a basis equivalent to services rendered." When they were sure that a man was in a position to pay, he should be made to pay the same as he would pay outside.

Dr. Clarke enquired whether the doctors should be paid too. To this the Premier replied that no payment should be made to the medical officer in charge of the institution. Dr. Clarke retorted that the honorary medical officers got nothing for doing work in the hospital. He considered that this was a sore point, and Dr. Sprott held that they were cutting their own throats.

The Premier admitted that it had become a very difficult question. As far as the question of the management of the hospital generally was concerned, that had been dealt with by the Government, but owing to pressure of business it would be absolutely impossible to introduce a Bill into

Parliament during the present session. He could only promise to go carefully into the matter during the recess, and to consider the Bill from the point of view of trying to meet the wishes of the members of the deputation in regard to putting the hospitals on a better footing. Whether they could go the whole way was a matter which would have to be considered by the Government. It seemed to him that the only logical conclusion was to prohibit the payment of fees altogether, and that opened up a very serious financial aspect.

Dr. Clarke thanked the Premier for having received the deputation, and expressed his regret that he had not been able to satisfy the members on the chief point. He was afraid that the reply would leave the medical profession in a very uncomfortable condition of unrest.

Public Health.

THE HEALTH OF NEW SOUTH WALES.

The following notifications have been received by the Department of Public Health, New South Wales, during the week ending November 25, 1916:—

	Metropolitan District.		Hunter River District.		Rest of State.		Total.	
	Cs.	Dths.	Cs.	Dths.	Cs.	Dths.	Cs.	Dths.
Enteric Fever ..	17	2	3	0	13	2	33	4
Scarlatina ..	108	1	12	0	16	0	136	1
Diphtheria ..	65	2	2	0	35	0	102	2
C'bro-Sp'l Menin.	4	3	0	0	1	1	5	4
Poliomyelitis ..	0	0	0	0	1	0	1	0
* Pul. Tuberculosis	26	14	0	2	19	1	45	17
Malaria ..	1	0	0	0	1	0	2	0

* Notifiable only in the Metropolitan and Hunter River Districts, and, since October 2, 1916, in the Blue Mountain Shire and Katoomba Municipality.

THE HEALTH OF VICTORIA.

The following notifications have been received by the Department of Public Health, Victoria, during the week ending November 26, 1916:—

	Metropolitan.		Rest of State.		Total.	
	Cs.	Dths.	Cs.	Dths.	Cs.	Dths.
Diphtheria ..	35	1	30	1	65	2
Scarlatina ..	18	0	6	0	24	0
Enteric Fever ..	3	0	6	0	9	0
Pulmonary Tuberculosis	48	4	16	8	64	12
C'bro-Spinal Meningitis	7	—	2	—	9	—

INFECTIVE DISEASES IN QUEENSLAND.

The following notifications have been received by the Department of Public Health, Queensland, during the week ending November 25, 1916:—

Disease.	No. of Cases.
Scarlatina ..	19
Pulmonary Tuberculosis ..	11
Enteric Fever ..	23
Diphtheria ..	46
Puerperal Fever ..	1
Malaria ..	6
Ankylostomiasis ..	1
Erysipelas ..	1
Cerebro-Spinal Meningitis ..	1
Anterior Poliomyelitis ..	1

THE HEALTH OF WESTERN AUSTRALIA.

The following notifications have been received by the Department of Public Health during the fortnight ending November 18, 1916:—

Disease.	Metropolitan Cases.	Rest of State Cases.	Totals Cases.
Enteric Fever ..	6	6	12
Diphtheria ..	17	3	20
Scarlatina ..	4	1	5
Pulmonary Tuberculosis ..	10	8	18
Cerebro-Spinal Meningitis ..	2	4	6
Erysipelas ..	1	0	1
Pyæmia ..	1	0	1
Septicæmia ..	2	0	2
Malaria ..	2	0	2
Beri-beri ..	0	2	2
Ophthalmia Neonatorum ..	1	0	1

THE HEALTH OF TASMANIA.

The following notifications have been received by the Department of Public Health, Tasmania, during the week ending November 25, 1916:—

Disease.	Hobart. Cases.	Launceston. Cases.	Country. Cases.	Whole State. Cases.
Diphtheria	1 ..	4 ..	13 ..	18
Pulmonary Tuberculosis	0 ..	0 ..	1 ..	1
Scarlatina	0 ..	0 ..	1 ..	1
Enteric Fever	0 ..	0 ..	1 ..	1
C'bro-Spinal Meningitis	1 ..	0 ..	0 ..	1
Puerperal Fever	0 ..	0 ..	1 ..	1

THE HEALTH OF SOUTH AUSTRALIA.

The following notifications have been received by the Central Board of Health, South Australia, during the fortnight ending November 18, 1916:—

Disease.	Adelaide. Ca. Dths.	Rest of State. Ca. Dths.	Totals. Ca. Dths.
Morbili	36 1 .. 146	0 .. 182	1
Pertussis	16 1 .. 102	6 .. 117	7
Pulmonary Tuberculosis	3 5 .. 12	14 .. 15	19
Diphtheria	8 4 .. 24	2 .. 32	6
Enteric Fever	2 0 .. 10	0 .. 12	0
Scarlatina	0 0 .. 6	0 .. 6	0
C'bro-Spinal Meningitis	1 1 .. 3	1 .. 4	2
Puerperal Fever	0 1 .. 3	2 .. 3	3
Erysipelas	2 0 .. 0	0 .. 2	0

Medical Appointments.

Dr. G. T. Woolley has resigned his position as Officer of Health at Castlemaine, Victoria. Dr. A. M. Hill has been appointed in his stead.

Dr. H. G. Loughnan has been appointed Officer of Health for Kyneton, Carlisle, Lauriston, Edgecumbe and Tylden Ridings, Victoria, a vacancy created by the death of Dr. G. O. Rigby.

Dr. R. J. Fullerton has been appointed Health Officer for Narracan Shire, in place of Dr. R. C. Marsden (resigned). Dr. R. Salts has been appointed Officer of Health for the Western portion of Towong Shire, Dr. H. F. Wickens having resigned.

Dr. Francis Frederick Brown has been appointed Government Medical Officer at Bourke. This position was held until recently by Dr. R. Dey.

Dr. Cyril Howard Shearman, Perth, has been appointed a member of the Foods Standards Advisory Committee, under the provisions of "The Health Act, 1911-15."

Dr. P. H. Nutting has resigned his position as Resident Medical Officer at Fremantle Hospital as from the 31st October, 1916.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xxi.

Children's Hospital, Perth; Resident Medical Officer.

Medical Appointments.

IMPORTANT NOTICE.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429 Strand, London, W.C.

Branch.

VICTORIA.
(Hon. Sec., Medical Society Hall, East Melbourne.)

APPOINTMENTS.

Brunswick Medical Institute.
Bendigo Medical Institute.
Prahran United F.S. Dispensary.
Australian Prudential Association Proprietary, Limited.
National Provident Association.
Life Insurance Company of Australia, Limited.
Mutual National Provident Club.

Branch.

SOUTH AUSTRALIA.

(Hon. Sec., 3 North Terrace, Adelaide.)

QUEENSLAND.

(Hon. Sec., B.M.A. Building, Adelaide Street, Brisbane.)

WESTERN AUSTRALIA.

(Hon. Sec., 230 St. George's Terrace, Perth.)

APPOINTMENTS.

The F.S. Medical Assoc., Incorp., Adelaide.

Brisbane United F.S. Institute

Swan District Medical Officer.
All Contract Practice Appointments in Western Australia.

Department of Public Instruction—Appointments as Salaried Medical Officers, with duties which include the treatment of school children.

Australian Natives' Association.
Balmain United F.S. Dispensary.
Canterbury United F.S. Dispensary.
Leichhardt and Petersham Dispensary.
M.U. Oddfellows' Med. Inst., Elizabeth Street, Sydney.

Marrickville United F.S. Dispensary.
N.S.W. Ambulance Association and Transport Brigade.

North Sydney United F.S.
People's Prudential Benefit Society.
Phoenix Mutual Provident Society.
F.S. Lodges at Casino.
F.S. Lodges at Lithgow.
F.S. Lodges at Orange.
F.S. Lodges at Parramatta, Penrith, Auburn, and Lidcombe.
Newcastle Collieries — Killingworth, Seaham Nos. 1 and 2, West Wallsend.

NEW ZEALAND:
WELLINGTON
DIVISION.

(Hon. Sec., Wellington.)

F.S. Lodges, Wellington, N.Z.

Diary for the Month.

- Dec. 12.—N.S.W. Branch, B.M.A., Executive and Finance Committee.
Dec. 12.—Tas. Branch, B.M.A., Council and Branch.
Dec. 13.—South Sydney Med. Assoc. (N.S.W.).
Dec. 14.—Vic. Branch, B.M.A., Council.
Dec. 15.—N.S.W. Branch, B.M.A., Branch (Ordinary).
Dec. 15.—Eastern Suburbs Med. Assoc. (N.S.W.).
Dec. 19.—N.S.W. Branch, B.M.A., Medical Politics Committee.
Dec. 21.—City Med. Assoc. (N.S.W.).
Dec. 22.—Q. Branch, B.M.A., Council.
1917.
Jan. 9.—N.S.W. Branch, B.M.A., Council (Quarterly).

EDITORIAL NOTICES.

Manuscripts forwarded to the office of this Journal cannot under any circumstances be returned.
Original articles forwarded for publication are understood to be offered to *The Medical Journal of Australia* alone, unless the contrary be stated.
All communications should be addressed to "The Editor," *The Medical Journal of Australia*, B.M.A. Building, 30-34 Elizabeth Street, Sydney, New South Wales.